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JOURNAL OF NEUROLOGY NEUROSURGERY AND PSYCHIATRY

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LONDON
BRITISH MEDICAL ASSOCIATION
TAVISTOCK SQUARE, W.C.1

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Brown, A. B. (1845). *Rev. Neurol.*, 169, 427.

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J. Neurol. Neurosurg. Psychiat., 1952, **15**, 1.

POSTERIOR FOSSA DERMOID CYSTS WITH SPECIAL REFERENCE TO INTRACRANIAL INFECTION

BY

VALENTINE LOGUE and KENNETH TILL

From the Department of Neurological Surgery, St. George's Hospital, London

The full understanding of the natural history of intracranial dermoid cysts has long remained incomplete owing to the infrequent occurrence of these tumours, but of recent years a sufficient number of isolated case reports have appeared to explain many of their obscure but interesting features. In particular these reports have revealed that dermoid cysts lying in the posterior fossa are prone to certain complications, notably infective, and have signs both clinical and radiological which are peculiar to their situation and which are not shared by the supratentorial dermoids.

Some aspects of dermoid cysts are well known. They arise from misplacement of fragments of ectoderm in the vicinity of the developing neural tube about the third or fourth week of embryonic life. Their derivation from skin is amply confirmed by microscopical examination of the cyst wall, which is seen to be composed of corium and epidermis in which appear some or all of the structures present in normal skin ; sebaceous glands and sweat glands which may be well formed or only rudimentary, hair and hair follicles, fat, muscle, elastic fibres, and blood vessels. This complete skin layer, however, is variable in its extent and may be confined to a small area of the cyst wall, the remainder being lined solely by epidermis.

The contents of the cyst comprise fat cells, sebaceous material and cholesterol, with usually a number of long, coiled hairs lying separately or in clumps, and which at operation form the diagnostic feature of this type of tumour. An individual cyst may be round, oval, or bilocular, the shape depending to some extent on its situation, and in size varies from a few millimetres to several centimetres in diameter.

Dermoid cysts may occur anywhere within the skull, but their commonest single site is in the posterior fossa at or near the midline, where approximately one-third of the total number of recorded cases have been located (Brock and Klencke, 1931 ; Sweet, 1940).

It can be generally stated that whatever its intracranial situation a dermoid cyst tends slowly to enlarge as a result of the secretion of sebaceous material and the desquamation of epithelium within it, and that after a varying time symptoms are produced resembling those arising from the more slowly growing types of brain tumour. Occasionally the symptoms may be rapidly progressive or, on the other hand, the cyst may enlarge so slowly that it does not manifest its presence during the course of a long life.

Dermoid cysts lying adjacent to the ventricles or subarachnoid space carry a further hazard in that they may rupture and discharge their contents into the cerebrospinal fluid to produce symptoms bizarrely various. One clinical picture is that of an acute cerebral vascular accident resulting in coma and death within a few hours or days, the diagnosis being revealed by the oily fluid obtained at spinal puncture. Another syndrome resembles subacute meningitis which may run a course of several weeks before terminating in spontaneous recovery or death. A rarer manifestation of rupture is that of progressive internal hydrocephalus without symptoms of meningitis. This is the result of a diffuse hypertrophy of ependymal and leptomeningeal cells around deposits of lipoid, which leads to obstruction of the flow of cerebrospinal fluid through the aqueduct of Sylvius and over the convexity of the brain. It has been shown experimentally that the cholesterol in the cyst contents is the substance responsible for these meningeal reactions (Bender, 1925 ; Greenfield, 1932).

Metastatic blood stream infection from a focus elsewhere in the body is another rare complication. Among our own cases of posterior fossa dermoid cysts there is one in an 11-day-old infant which was infected as part of a pyaemia from umbilical cord sepsis.

These manifestations and complications are common to dermoid cysts whatever their intracranial situation, whether above or below the

tentorium, but those situated in the posterior fossa possess in addition certain features which, while making their clinical diagnosis more evident, add considerably to the danger of their presence within the skull.

Posterior Fossa Dermoid Cysts

The most notable characteristic of dermoid cysts in the posterior fossa is their tendency to lie in the midline of the skull whatever their relationship to the intracranial structures, whether outside or within the dura mater, and although in the latter position they may enlarge asymmetrically over one or other cerebellar hemisphere the main bulk of the tumour overlies the vermis or outlet of the fourth ventricle. This constant site is probably related to the development of the tentorium and falx cerebelli which occurs as an invagination of two folds of dura, strictly in the midline of the posterior fossa, and may draw in fragments of the skin layer with it. Confirmation is lent to this by the observation that the intradural cysts commonly have a firm attachment to the tentorium. Exceptionally a cyst may lie at a distance from the midline, in the cerebello-pontine angle or adjacent to the brain stem, but such deviations from the usual position are extremely rare.

Another peculiarity of these tumours is that the attachment to the skin from which they develop may be retained as a narrow sinus lined with epithelium, which links the cyst with the skin through an opening in the occipital bone. This is a feature which distinguishes them from the supratentorial dermoid cysts, where a primary skin attachment does not occur and which allies them developmentally with the dermoid cysts and congenital dermal sinuses occurring in the spinal canal. This connexion with the skin, perhaps better called the occipital dermal sinus, will permit extrusion of the dermoid contents to the exterior and, what is more important, will provide direct access to the cavity of the tumour for any bacteria lying on the surface. If the tumour should lie intradurally the invading organisms, once having entered the cyst, are separated from the cerebrospinal fluid only by the cyst lining and pia arachnoid, a tenuous barrier which may offer little resistance to the further spread of infection.

A study of the reported cases of posterior fossa dermoid cysts reveals that they can be divided into four groups depending on two criteria ; first their anatomical situation, whether extradural or intradural, and secondly the degree of development of the occipital dermal sinus, whether absent altogether, partial or complete. Furthermore it is found that with cysts lying extradurally the occipital dermal sinus seems always to persist in its entirety, linking the tumour with the skin through an opening in the occipital bone. With the intradural dermoid cysts, however, there is a wider variation in the dermal sinus. In most cases the connexion to the skin disappears completely leaving a cyst in the depths of the posterior fossa with no abnormality of the overlying bone or skin to record its presence. In those remaining cases where the dermal sinus persists it does so in one of two forms, either as a complete sinus leading from tumour to skin in a fashion similar to the extradural variety, or merely as a short tube attached to the skin and ending blindly in the subcutaneous tissue over the occipital protuberance.

These four groups, one extradural and three intradural, can be set down thus : (1) extradural dermoid cyst with complete dermal sinus ; (2) intradural dermoid cyst (*with no dermal sinus*) ; (3) intradural dermoid cyst with an *incomplete dermal sinus ;* and (4) intradural dermoid cyst with a *complete dermal sinus.*

These variants are illustrated diagrammatically in Fig. 1 and the relative frequency with which they occur can be seen from Table 1, which is composed of 25 cases gathered from the literature in which the descriptive details are adequate, and seven cases of our own, a total of 32.

TABLE I

RELATIVE FREQUENCY OF FOUR TYPES OF POSTERIOR FOSSA DERMOID CYST IN 32 CASES

Type of Tumour	No. of Reported Cases*	No. in Present Series	Total
Extradural with complete sinus ..	2	1	3
Intradural without a dermal sinus ..	16	2	18
Intradural with incomplete sinus ..	3	1	4
Intradural with complete sinus ..	4	3	7
Total	25	7	32

*Bellani (1925). Clairat (1838) ; Courville and Kimball (1936) ; Downes (1929) ; Gladstone (1937) ; Gorog (1927) ; Horrax (1922) ; Irving (1879) ; Kruse (1891) ; Kwan (1910) ; Lannelongue (1910) ; Love and Kernohan (1936) ; Martin and Davis (1943) ; Miller (1937) ; Munro and Wegner (1937) ; Quade and Craig (1939) ; Rand and Reeves (1943) ; Ruschhaupt (1903) ; Sweet (1940) ; Todesco (1927) ; Verbiest and Zeldenrust (1938) ; Webster (1938).

Clinical Features of Individual Cysts

Extradural Dermoid Cyst with Complete Dermal Sinus.—This type of tumour lies outside the dura embedded in the midline of the occipital bone which it thins and erodes in a typical manner. The dermal

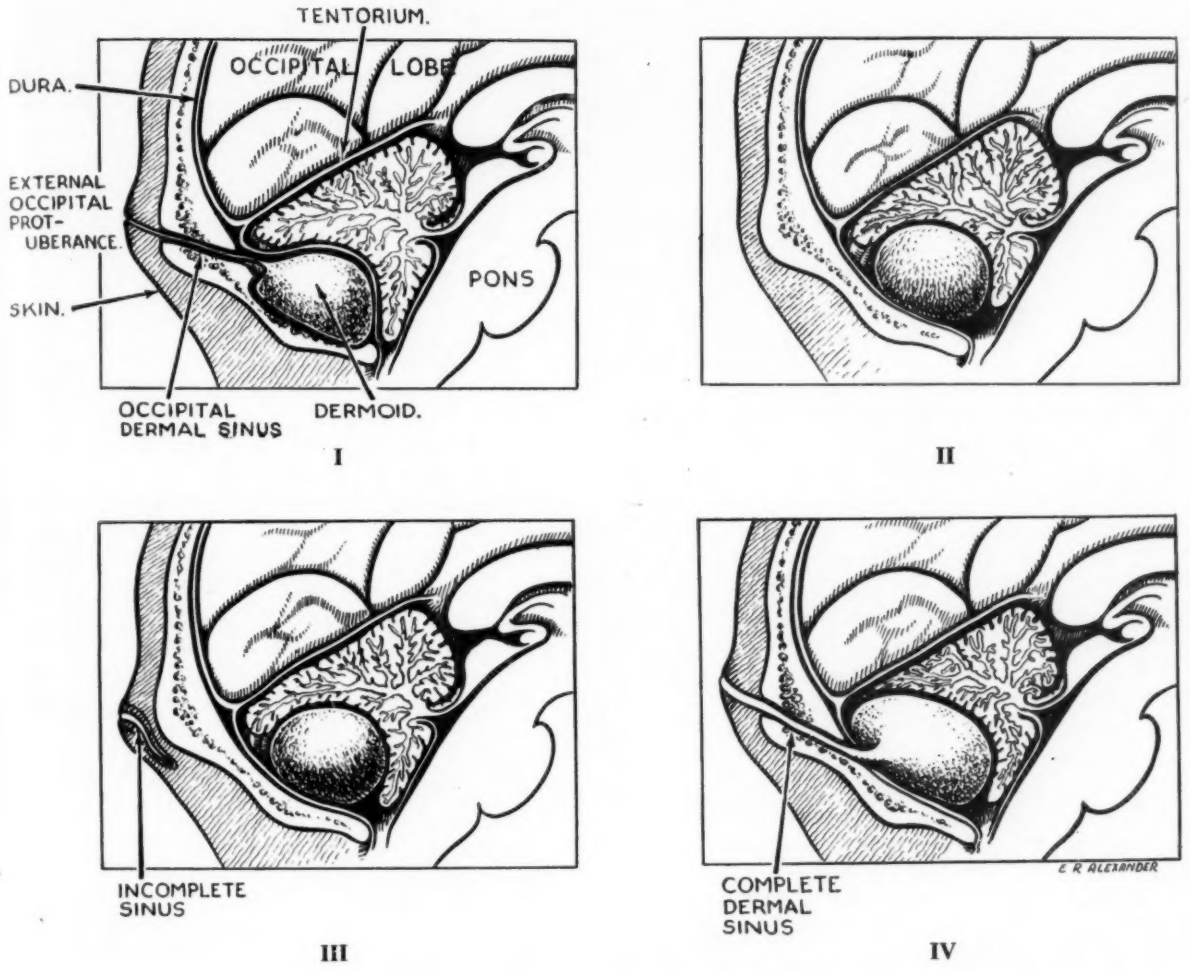


FIG. 1.—Semi-diagrammatic drawing of a sagittal section through the posterior fossa to illustrate the four varieties of posterior fossa dermoid cysts. I, Extradural dermoid cyst with complete dermal sinus; II, intradural dermoid cyst without a dermal sinus; III, intradural dermoid cyst with incomplete sinus; IV, intradural dermoid cyst with complete sinus.

sinus forms a fine stalk which arises from the upper pole of the tumour and pierces the remaining thickness of bone to gain attachment to the skin over the external occipital protuberance. The contents of the cyst pass along this stalk either to discharge on to the skin and so draw attention to its presence, or, if the exit is blocked, to collect in the terminal portion of the sinus and form a subcutaneous swelling, which is often removed surgically and mistakenly as a " sebaceous cyst ", the incision never healing afterwards.

The history in this variety of dermoid is quite characteristic and is illustrated by our first case.

Case 1 : B. B.—This was a boy aged 2 years. Shortly after birth it was noticed that he had a lump the size of a pea at the back of his head. This was removed at a local hospital when he was 11 months old and was said to have been a " cyst ". The incision did not close after the operation and discharged yellow fluid intermittently. At the age of 18 months and again at 2 years the sinus was re-excised, without success, at the same hospital. Shortly after the third operation the child was admitted to this department. Clinical examination revealed a small sinus over the occipital protuberance which discharged pus containing *Staph. aureus*. No abnormality could be detected in the central nervous system.

A radiograph of the skull (in the half-axial view) showed a roughly circular defect 1·5 cm. in diameter, surrounded by a smooth margin of sclerotic bone situated in the midline of the occiput just below the inion (Fig. 2).

At operation the sinus and surrounding scar tissue was explored, but no connexion could be found with the underlying bone and it was unfortunately again only locally excised. This time it remained healed for a few weeks before breaking down once more. The sinus

was then re-explored (for the fifth time), and on this occasion a small stalk was seen leading obliquely downwards to enter a channel in the external occipital protuberance. The shell of bone around this channel was removed to uncap a dermoid cyst containing yellow, cheese-like material, lying in contact with, but outside, the dura just below the torcula, with the dermal sinus

FIG. 2.—Radiograph (half-axial projection) of Case 1 showing the circular defect with sclerosed edge in the occipital bone.

entering at its upper pole. The capsule and contents were completely excised, after which the wound healed promptly and has remained so ever since.

Microscopical examination showed a lining of squamous epithelium bearing hair follicles and sebaceous glands (Fig. 3).

This case illustrates the significance of small " cysts " and sinuses over the midline of the occiput in children and the need for radiography of the bone in this region before contemplating surgical removal.

The *x*-ray appearance is unmistakable, although occasionally mimicked by the epidermoid tumour, and consists of a circular defect varying in different cases from 1·5 to 5 cm. in diameter, with a smooth, sometimes scalloped, margin of sclerotic bone, situated in the midline just below the inion. The small channel conveying the dermal sinus lies above the defect, but cannot as a rule be distinguished on account of its fine calibre and the fact that it passes obliquely through the thick bone of the external protuberance.

This variety of dermoid cyst typically does not produce intracranial symptoms, but occasional cases have been reported in which the extradural mass had enlarged sufficiently to compress the contents of the posterior fossa (Martin and Davis, 1943).

Intradural Dermoid Cyst without Dermal Sinus.—Table I shows this to be the commonest variety of posterior fossa dermoid. In general this type of cyst tends to manifest itself at a later age than those in which the dermal sinus persists and symptoms do not usually appear until adult life. It has no certain diagnostic features and, as its symptoms do not differ notably from those of other tumours of the cerebellum, they do not warrant a detailed description. There are certain points, however, which may arouse suspicion of its identity. These are (1) an extremely slow evolution of symptoms; (2) signs pointing to a predominantly midline situation of the tumour, in contrast to the frankly neoplastic cysts in adult life, which tend to lie more laterally, in the cerebellar hemispheres; (3) the demonstration by radiography of congenital anomalies of the cervical spine, in particular, fusion of the laminae or vertebral bodies.

When exposed at operation the tumour, blue or green, is seen to overlie the vermis pushing it upwards and forwards, indenting the adjacent surfaces of the cerebellar hemispheres and displacing the cisterna magna downwards.

Less frequently it lies at a lower level and projects into the fourth ventricle, or may even extend through the foramen magnum to produce additional symptoms of involvement of the spinal cord.

Intradural Dermoid with Incomplete Dermal Sinus.—In this variety the tumour lies in a similar situation

FIG. 3.—Section shows a well-preserved part of the cyst wall consisting of a deep collagen layer and well formed stratified squamous epithelium. Hair follicles and sebaceous glands are present. Haematoxylin and eosin. × 45.

to the previous one and produces identical neurological symptoms.

The dermal sinus originates from a dimple in the skin overlying the occipital protuberance and extends for a short distance into the subcutaneous tissue to end either blindly, or attached to the periosteum. The sinus, although carrying no added risk of complications, will be of help in revealing the nature of the underlying tumour. This occurred in one of our cases.

Case 2 : E. B.—This was a woman aged 61, who for one year had had symptoms referable to a midline posterior fossa tumour causing an internal hydrocephalus.

Radiography showed, in addition to signs of raised intracranial pressure, fusion of the bodies and laminae of the second, third, fourth, and fifth cervical vertebrae. There was no defect in the bone of the occiput.

Vertriculography confirmed the hydrocephalus and demonstrated an aqueductal " kink " characteristic of a large midline cerebellar tumour. When the head was shaved a dimple 2 mm. in diameter was seen lying over the occipital protuberance and a small cord-like process could be palpated running obliquely downwards and forwards from it for a short distance. The presence of a dermoid tumour had been suggested by the congenital deformity of the cervical spine, and the discovery of the dermal sinus made this diagnosis unequivocal. Such a tumour, the size of an egg, was found lying over the vermis and adjacent portions of the cerebellar hemispheres and was completely removed. The sinus extended from the skin for 2 cm. into the subcutaneous tissue and ended blindly.

Intradural Dermoid with Complete Dermal Sinus.—The characteristic feature of this type is the presence of a dermal sinus leading from the midline intradural cyst through an opening in the occipital bone to be attached to the skin. The sinus discharges either as a natural phenomenon or, if its outlet is blocked, as a result of the surgical removal of its terminal dilated portion in the belief that it is a " sebaceous cyst ". In addition to the intermittent discharge of sebaceous material, which may be extremely variable in amount, the sinus provides a pathway whereby organisms can gain access to the intradural tumour and thence to the subarachnoid space and cerebellum.

We have three cases which illustrate the serious complications resulting from the entry of organisms into this variety of dermoid cyst.

Case 3 : G. R.—This was a girl of 4 years of age who, some three months before admission to this department, had become drowsy and irritable, complained of severe headache, and was shortly admitted to a local hospital in a semi-comatose condition. On spinal puncture a milky fluid was obtained from which *Bact. coli* was cultured. She was treated with systemic and intrathecal penicillin (streptomycin was not at that time available). The meningeal infection was overcome by the nineteenth day, but a raised pressure in the cerebrospinal fluid persisted with a slightly increased white cell count. During the fourth week after admission there was another episode of drowsiness, irritability, neck stiffness, and fever, but these signs subsided spontaneously and she was sent home three weeks later apparently well, except that she was unsteady on her feet and tended to fall if unsupported.

Two weeks elapsed and the child then began to vomit after her meals and again became drowsy and complained of headache. At this stage she was readmitted to the local hospital where examination revealed early papilloedema and a left lower facial weakness. A small fluctuant swelling had appeared over the occipital protuberance which was later incised releasing pus containing *Bact. coli.*

The patient was then transferred to this department. On examination, papilloedema was well marked in both eyes. The cranial nerves were otherwise normal, except for dilated, inactive pupils. The limbs on both sides showed an ataxia of cerebellar type with reduction of all tendon reflexes. The plantar responses were flexor. The child could walk only when supported and then very unsteadily, and if support was withdrawn she fell over.

Radiographs of the skull showed diastasis of the coronal and sagittal sutures. In the half axial projection a narrow channel 1 cm. in length with sclerosed walls leading down to an oval groove was seen in the occipital bone below and slightly to the left of the internal protuberance (Fig. 4). The significance of these findings was not appreciated at that time.

The diagnosis of a midline cerebellar abscess was made and the location confirmed by ventriculography. A needle introduced through a suboccipital burr hole entered an abscess from which 15 ml. of pus were aspirated and 20,000 units of penicillin and 2 ml. of thorotrast replaced. The infecting organism was identified as *Bact. coli.* The thorotrast outlined the abscess showing it to be irregularly spherical and measuring 3 cm. in diameter (Fig. 5). During the ensuing three and a half weeks serial radiographs showed that the abscess had grown bigger, and it was observed that the capsule did not take up the thorotrast in the manner usually seen in a brain abscess. It was decided to perform a total excision.

At operation a firm cord of tissue was found which ran from a skin dimple close to the scar over the occipital protuberance, obliquely down through a small channel in the bone to fuse with the dura just below the torcula. When this cord was cut across, pus and a number of long golden hairs extruded, which revealed the true nature of the tumour. The dura was then opened to expose a pus-filled dermoid cyst the size of an egg overlying the vermis and separating the hemispheres ; this was removed with considerable difficulty owing to numerous adhesions. The dermal sinus entered at its upper pole and its lower extremity was attached by a strand of tissue to the medulla at the lower end of the fourth ventricle.

Post-operatively sulphonamides and penicillin were

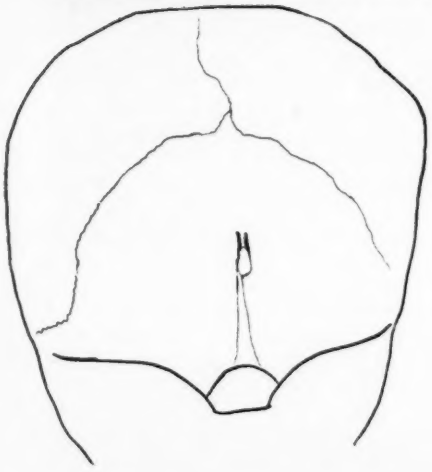


FIG. 4.—Radiograph and line drawing to show the channel and groove in the occipital bone in Case 3.

FIG. 5.—Radiograph of the skull of Case 3 showing the abscess cavity outlined by thorotrast. At the upper margin of the abscess the dense, bony walls of the channel conveying the dermal sinus can be seen.

continued and despite coliform bacilli being cultured from the ventricular fluid on two occasions, and the occurrence of a series of cerebellar fits, the child slowly recovered. She returned home four weeks after operation, by which time the papilloedema and all signs of cerebellar dysfunction had disappeared. She has been seen at intervals during the past three years and appears to be developing as a normal child.

Microscopical examination of the tumour confirmed the typical features of an infected dermoid cyst.

The evolution of the infection in this case can be divided into three stages. First the entrance of *Bact. coli* into the dermal sinus leading to an infection of the cyst, with presumably an immediate spread to the leptomeninges. (It should be noted that there had never been sufficient discharge from the sinus to draw the parents' attention to it, and intracranial infection was the first sign of the presence of the tumour.) This initial attack of meningitis, and a second milder episode three weeks later, was overcome, but infection in the cyst progressed, converting it into an abscess which then produced all the signs of a midline expanding lesion. Also during this phase some pus tracked externally along the dermal sinus to collect under the scalp. Finally, the intracranial abscess increased in size despite aspiration and the introduction of an antibiotic and this, combined with the unusual behaviour of the thorotrast suggesting an atypical pathology, determined its surgical removal.

This case emphasizes the rule that the occurrence of what appears to be a primary coliform meningitis should demand a careful search for a " sebaceous cyst," dimple, or sinus in the skin of the occiput (and of course the spine). The case also demonstrates the radiological features which we have come to regard as diagnostic of this variety of dermoid cyst, consisting of a small channel with dense walls leading down to an oval groove in the midline of the occipital bone just below the inion. This appearance has to be distinguished from other openings in the bone in this situation and the main points of differential diagnosis will be mentioned later.

Case 4 : J. S.—This was a girl aged 16 months at her first admission to this department, who, shortly after birth, was noted to have a small swelling over the centre of the occiput which about once a month discharged some brownish material and then closed up. This lump had been excised two weeks earlier at another hospital, when a small cord was found leading down to a slit-like opening in the bone of the occipital protuberance. As this stalk was tied off it was observed to contain some fine black hairs. The wound did not heal and the child was then transferred to this department.

Examination revealed a small scab in the centre of an otherwise healed occipital incision. There was no abnormality detected in the central nervous system.

A radiograph of the skull in the half axial view showed, as in the previous case, a narrow channel in the midline of the occipital bone leading down to an oval groove

(Fig. 6). An air study was performed to determine whether there was an intradural extension of the tumour and this showed a ventricular system of normal size, with no deformity of the aqueduct or fourth ventricle. It was believed that these findings excluded the presence of an intradural tumour of any extent and it was thought safe to postpone surgery until the patient was older. The dermal sinus closed at this time and the child returned home.

She was seen three months after leaving hospital and again 12 months later, when she seemed perfectly well, and, as the sinus had remained healed, nothing further was done about the removal of the dermoid cyst.

The child was admitted as an emergency 20 months after leaving hospital. During the preceding four months she had experienced several attacks, lasting from 12 to 36 hours, of right occipital headache, associated with profuse vomiting and neck stiffness. Two days before admission a further attack occurred, after which she improved for a few hours and then rapidly became drowsy with a temperature of 101·4°F and had two generalized convulsions. On examination, the child was drowsy, but accessible. There was marked neck retraction and the skull had a boxy note on percussion. The occipital incision and dermal sinus were firmly healed. There was no papilloedema and the remainder of the nervous system disclosed no abnormality other than a positive Kernig's sign.

Ventricular puncture was performed and fluid under high pressure was withdrawn which contained 400 polymorphs per c.mm. and organisms identified as *Staph. aureus* (coagulase positive), which were sensitive to penicillin.

Despite intensive antibiotic therapy the patient's condition deteriorated, with a rising temperature and respiration rate and she died two days later. At necropsy a diffuse meningitis was revealed, the subarachnoid space being filled with thick, yellow exudate. A dermoid cyst measuring 3 × 2 × 2 cm., distended with pus and containing many long, golden hairs, was found lying between the cerebellar hemispheres. Attached to its upper pole was the dermal sinus which passed through a defect in the occipital bone to fuse with the skin (Figs. 7 and 8).

The course of the infection in this case can be divided into four phases : (1) organisms entered the cyst during one of the episodes when the sinus was discharging ; (2) the sinus then healed and the infection remained quiescent for 16 months, with the child apparently perfectly well ; (3) the organisms then assumed a more pathogenic character converting the cyst into an abscess with a resultant rise in intracranial tension and recurring attacks of headache and vomiting over four months ; (4) infection finally spread to the meninges with a fulminating meningitis.

Bearing in mind the lessons learned from these two cases, we decided that when next a patient was seen with an occipital dermal sinus in conjunction with the typical radiographic appearances in the occiput indicating an intracranial extension, the dermoid cyst should be removed forthwith before the development of inflammatory complications.

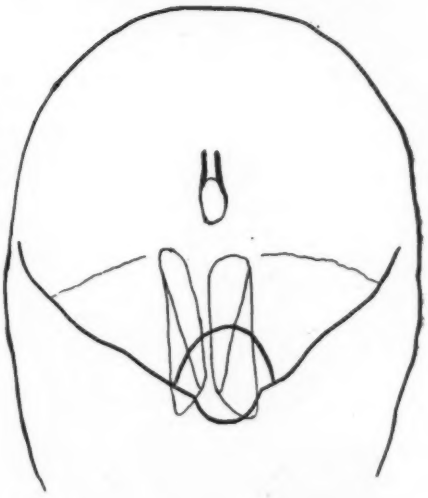


FIG. 6.—Radiograph (half-axial view) and line drawing to show the channel and groove in the occipital bone in Case 4. (A ventriculogram has been performed and air can be seen in the frontal horns and bodies of the ventricles.)

The following case was referred for treatment.

Case 5 : J. W.—This was a boy aged 11 months. Shortly after birth the mother noticed a pin-point opening at the back of the child's head. When 4 months old some fluid, which matted the child's hair, discharged from the opening. At 5 months of age and again at 9 months further discharges occurred, after which the sinus had remained healed for the two months before admission. There had been no intracranial symptoms.

Examination disclosed a soft swelling 2 cm. in diameter lying just above the occipital protuberance and bearing on its surface a dimple measuring 2 mm. across (Figs. 9 and 10). On rolling the skin the dimple retracted further, indicating a deep attachment, and a firm cord could be felt leading from it down to the bone. The punctum was healed and no sebaceous material

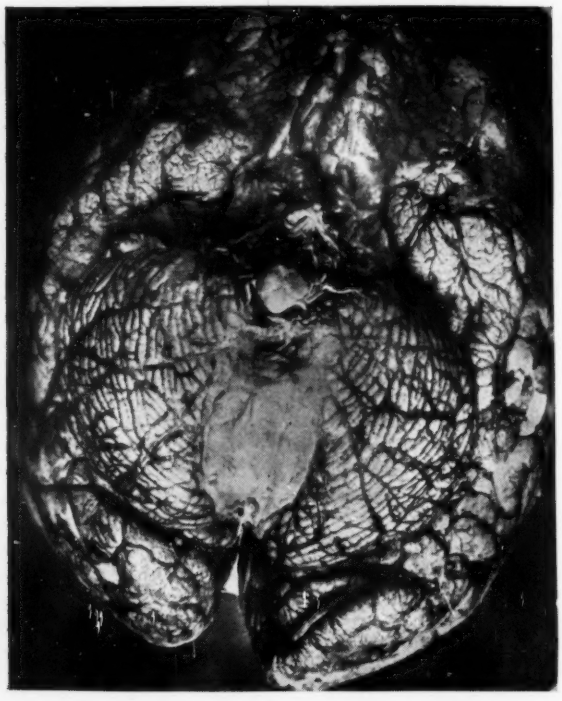
could be expressed from it. There were no abnormal signs in the central nervous system.

Radiographs of the skull in the half axial projection showed in the midline below the internal protuberance the typical short channel and groove (Fig. 11). In an attempt to ascertain the size of the intracranial portion of the dermoid, an encephalogram was performed which showed a normal cisterna magna, fourth ventricle, and aqueduct (Fig. 12). The cerebrospinal fluid obtained during this procedure was normal in all respects.

At operation, through a vertical incision, the punctum in the skin was encircled and the dermal sinus exposed. This ran obliquely downwards for 2 cm. to enter a slit-like channel in the bone just below the occipital protuberance (Fig. 13). The bone surrounding the channel was removed to reveal the inner end of the sinus which expanded in trumpet-like fashion, occupying the groove seen in the radiograph, and fused with the dura round the margins of the posterior surface of the intradural dermoid (Fig. 14). An incision was made in the dura mater to encircle this attachment. Projecting into the cisterna magna from the inner surface of the dura was a thin-walled, yellow dermoid cyst measuring 1 × 1·5 cm., its upper pole being adherent to the tentorium in the midline. This latter attachment, containing several large veins, was divided and the dermoid with the dermal sinus could then be lifted out (Fig. 15). Its only connexion with the cerebellum was by a small artery running to it from the vermis.

Some of the contents of the tumour were removed for culture as soon as it had been excised and a profuse growth of *B. proteus* resulted.

FIG. 7.—Photograph of base of brain of Case 4. The collapsed dermoid is seen overlying the vermis and outlet of the fourth ventricle. Some hairs are protruding from an opening in its upper pole where the dermal sinus was attached.



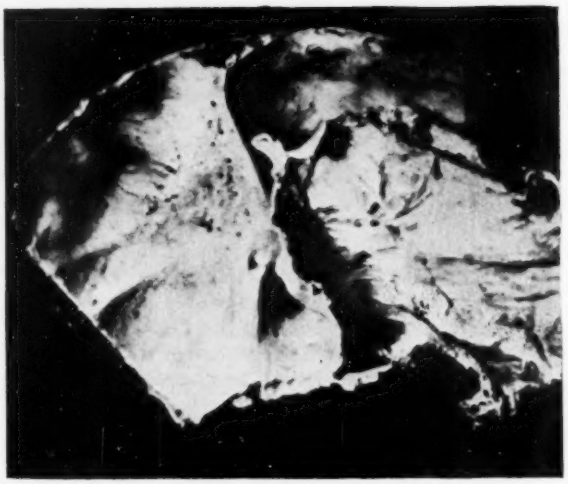


FIG. 8.—Photograph of the central portion of the occipital bone in Case 4 from which the dura has been partly stripped. The groove with its margins converging to enclose the opening of the channel is seen in the lower part of the bone.

Forty-eight hours after the operation the child developed a temperature of 104°F. and became restless and irritable. Spinal puncture disclosed a cloudy fluid containing 5,500 polymorphs, and culture grew the same *B. proteus* as found in the dermoid, which was sensitive to streptomycin. The child was treated with systemic and intrathecal streptomycin and the meningitis was cured in 12 days. He was sent home five weeks after operation and has remained perfectly well ever since (one year).

There are several points of resemblance between this and the previous case. Both presented in the same manner with a discharging dermal sinus. Both showed the pathognomonic *x*-ray change in the occipital bone, and careful air studies failed to demonstrate the presence of a tumour in the posterior fossa. The sinus remained healed, in the first patient for 16 months, and we inferred that the intracranial infection was due to the activation of organisms which had gained entry at a much earlier stage when the tumour was discharging and had then remained quiescent. This inference that pathogenic organisms could remain inactive in these dermoid cysts for long periods was amply confirmed by the second patient, when organisms were actually found in the cyst two months after the sinus had last discharged and a brisk meningitis left no doubt as to their pathogenicity.

Discussion

Symptomatology.—In the case of the intradural dermoid (*a*) with no dermal sinus, or (*b*) with an incomplete sinus, the cyst enlarges slowly and manifests itself in adult life with the symptoms of a midline cerebellar tumour, when it can be removed

FIG. 10.—The dimple and dermal sinus in Case 5 leading down to the small bone defect have been outlined with a skin pencil.

FIG. 9.—Photograph showing the skin dimple over the external occipital protuberance in Case 5.

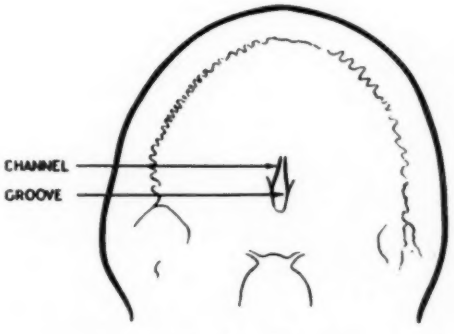


FIG. 11.—Radiograph and line drawing to show the groove and channel in the occipital bone in Case 5.

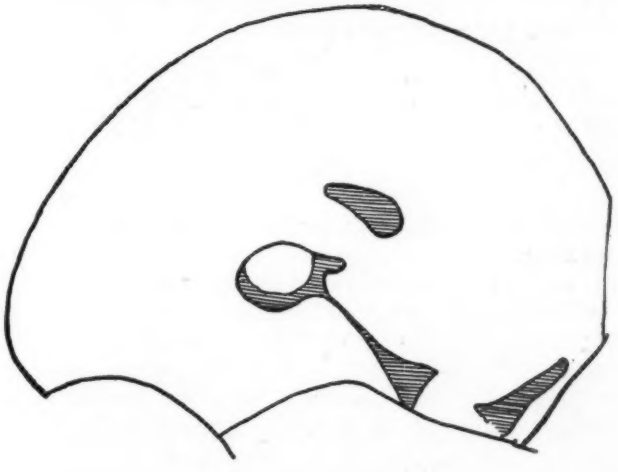


FIG. 12.—Tracing of encephalogram demonstrating the normal appearance of the cisterna magna, aqueduct, and fourth ventricle.

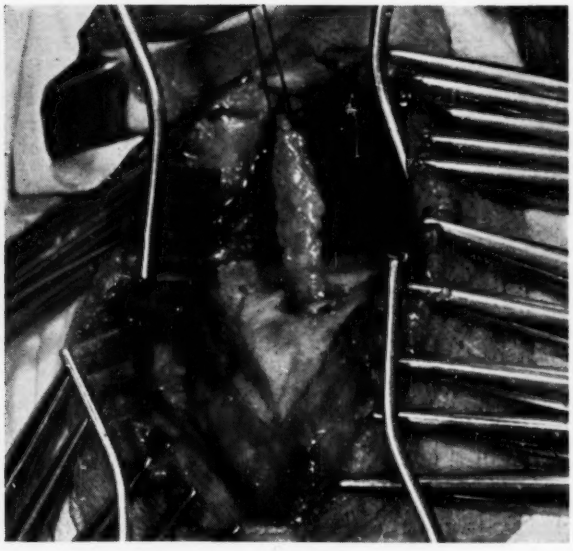


FIG. 13.—Photograph during operation on Case 5. The vertex of the child's head is towards the lower border of the photograph. The dermal sinus has been dissected out and then rotated caudally to expose its neck running into the bone.

FIG. 14. Photograph during operation on Case 5. The bone has been removed to expose the dura of the posterior fossa and the dermal sinus has been rotated over to the patient's right. The trumpet-like expansion of the dermal sinus as it fuses with the posterior border of the dermoid is well shown.

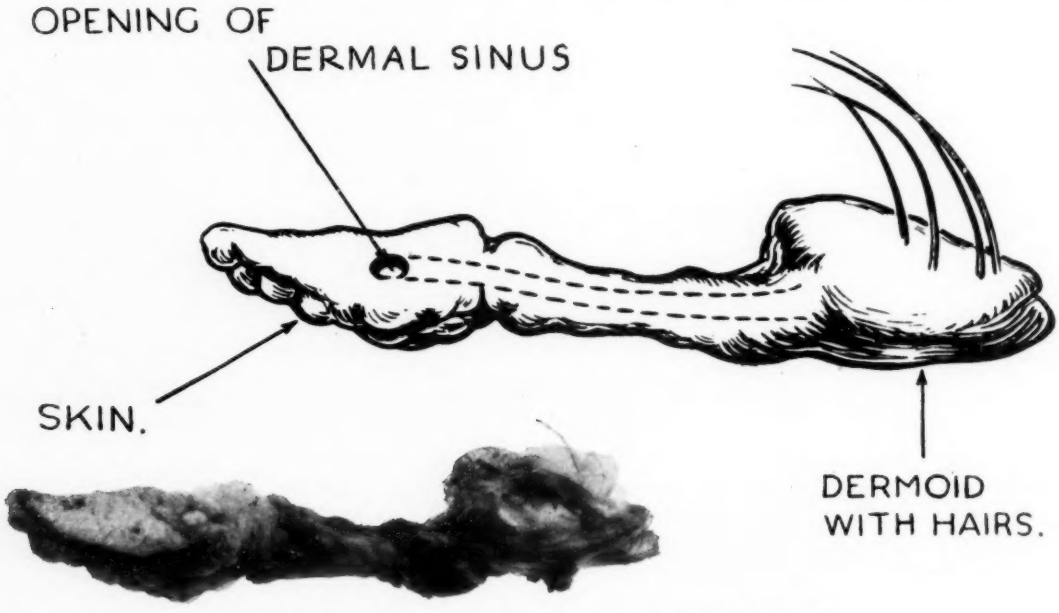


FIG. 15.—Photograph and line drawing of excised dermoid in Case 5.

without much difficulty and with an excellent prognosis.

It is the presence of the complete dermal sinus which alters the mode of presentation and the prognosis. In the case of the *extradural* tumour with complete sinus the outlook is not so grave, and except for the rare occasions when the tumour enlarges sufficiently to encroach on the capacity of the posterior fossa it will merely present as a midline occipital sinus which discharges from time to time. A radiograph will demonstrate the typical bone change, and its surgical removal is simple.

It is when the intradural type of dermoid cyst retains its complete dermal sinus that the most serious results of this link with the skin are seen, and the fact that most of the reported cases, and

our own three examples, have occurred in infants or young children indicates that these tumours are vulnerable to infection at an early stage.

The first intimation of the presence of this type of dermoid cyst is usually that of a discharge of sebaceous contents from the occipital dermal sinus or the appearance of a subcutaneous swelling over the occipital protuberance. This swelling is often removed surgically so that a sinus appears secondarily. The discharge occurs intermittently, often at long intervals, but, as shown by Case 4, it is possible for septic complications to occur after the dermal sinus has been healed for a considerable time.

Once infection becomes active the subsequent course is variable. It may remain for a time confined to the cyst, converting it into a midline cerebellar abscess, but spread to the leptomeninges eventually occurs to cause a terminal meningitis (Case 4).

In other cases there is an early spread to the subarachnoid space, and if the child survives this attack of meningitis, then enlargement of the cyst as a midline abscess appears subsequently with or without further episodes of meningitis. A less frequent method of spread is that directly into the substance of the cerebellum to form a true cerebellar abscess, the dermoid cyst tending to remain small. This occurred in a case reported by Gladstone (1937).

In other patients where the discharge has not been sufficient to draw attention to the presence of the sinus, as in our Case 3, the first symptom will be referable to the intracranial spread of infection, either an abscess or meningitis, and it is in this type of case that the routine careful search of the occiput and spine for a dermal sinus may be so rewarding in diagnosis.

Diagnosis.—In cases where an occipital dermal sinus is suspected a search will reveal a dimple in the skin 1 or 2 mm. in diameter, often lying on top of a small subcutaneous swelling. The dimple is situated at a level just above the external occipital protuberance more or less in the midline and the dermal sinus can be palpated as a firm cord running from it obliquely down towards the occipital bone for a distance of about 2 cm.

The radiographic appearances of the extra- and intradural dermoid cysts with complete dermal sinus are characteristic. The appearance of the extradural variety has been described with Case 1. It may resemble that of a suboccipital meningocele, although clinical examination should leave little room for confusion, but its main points of difference are (1) that it tends to have a sclerotic margin, and (2) it erodes only the inner bone table and diploe, the outer table being preserved as a thin shell. The small opening for the dermal sinus, which would be a certain diagnostic feature, cannot as a rule be visualized in this type of case because of its very fine calibre.

The typical feature of the intradural dermoid cyst is that of a short channel leading down to an oval groove. The channel, transmitting the dermal sinus, passes obliquely through the bone and measures from 1 to 1·5 cm. in length and 3 to 6 mm. in width. The groove is on an average 1 cm. wide and 1 to 1·5 cm. long and accommodates the posterior aspect of the dermoid cyst and the expanded inner end of the sinus as it fuses with the tumour.

This radiographic appearance has to be distinguished from other openings in the midline, in particular those associated with a large occipital emissary vein or a suboccipital meningocele. The fundamental point of difference is that the channel and groove of the dermoid lie in the vertical plane and pass obliquely through the bone, so that they tend to be only faintly visible. In the other two conditions the foramina run horizontally backwards, so that a circular opening of varying size is produced, which is clearly punched out of the whole thickness of bone.

Air studies are of little value in ascertaining the presence of an intradural tumour before the onset of septic complications (Cases 4 and 5). The cysts at this stage are small, perhaps 1 to 1·5 cm. in diameter, and are not large enough to deform the aqueduct or fourth ventricle. It is only when the cyst is converted into an abscess many times greater than its original size that the typical deformity of a midline cerebellar expanding lesion is produced. In any event air studies are superfluous because the channel and groove in the occipital bone are irrefutable proof of the presence of the intradural dermoid cyst.

Management.—Problems of management arise only in the case of intradural cysts with complete dermal sinus. Even with this variety there is no great difficulty once septic complications have appeared, for the cyst must be removed as soon as the infection can be brought under control. It is when the condition has been diagnosed before the onset of intracranial infection (Case 5) that difficulties arise.

In our own case in which we performed a prophylactic removal, we had the humbling experience of causing the transfer of organisms from the cyst to the meninges and producing the very complication we were trying to avoid. However, we were able to isolate the infecting organism from the dermoid contents and ascertain its antibiotic sensitivity before the onset of the meningitis and then recog-

nize and treat the meningitis from its earliest stages, with a happy result.

On the other hand the dangers of postponing surgery until the spread of intracranial infection has occurred are well illustrated by Cases 3 and 4. In the former case excision of the tumour was carried out on a child already very sick, the technical removal was difficult owing to inflammatory adhesions, and the patient was critically ill afterwards. In the latter, despite the intensive use of an antibiotic, the meningitis could not be controlled and the child died before removal of the tumour was feasible.

These tumours obviously carry a serious risk whichever line of treatment is adopted ; whether it be early excision as a prophylactic against infection, or a removal delayed until intracranial spread of sepsis has occurred. However, although it may seem a drastic procedure to subject an infant or young child to a major intracranial operation when its only apparent disability is a discharging occipital sinus, the occurrence of serious inflammatory complications is inevitable sooner or later, and we believe that removal of the tumour without undue delay once the diagnosis has been made, and before septic complications occur, is the least dangerous method. The contamination of the meninges during the prophylactic excision of these dermoid cysts should not be unavoidable, for it is often possible to remove similar types of tumour from the lumbar portion of the spinal canal without producing meningitis, although it must be conceded that in this latter situation there is a greater tendency for the pia arachnoid to seal off the subarachnoid space round the cyst.

In any event it would probably be safer to anticipate post-operative meningitis, and in a future case we should give a full therapeutic course of the appropriate antibiotic once the organism had been isolated from the dermoid contents, and not wait for the development of meningitis. If it were possible, before operation, to identify the organisms from the discharge from the sinus, it would make for even greater safety.

Although posterior fossa dermoid cysts are uncommon tumours, they seem to occur more frequently than the paucity of recorded cases would suggest, in particular the variety of intradural dermoid with a complete dermal sinus, for the three cases reported here have come to this department in the last three and a half years. It is a condition which should be borne in mind when treating any infant or young child with symptoms of an abscess in the posterior fossa situated in the midline, or meningitis which is resistant to, or relapses despite adequate treatment, particularly if it is due to the coliform group of organisms.

Summary

A study of 32 cases of posterior fossa dermoid cysts reveals that four clinical types can be recognized depending on their anatomical situation and the presence or absence of an occipital dermal sinus.

The characteristic features of each type and their radiographic appearances are described.

Attention is drawn to the ease with which infection can enter the skull to cause meningitis or an abscess, in particular in the variety of intradural dermoid cyst with a complete dermal sinus.

It is a pleasure to acknowledge our indebtedness to Mr. Wylie McKissock for the records of Cases 1 to 4 ; to Professor T. Crawford for the pathological report and photomicrograph of the dermoid cyst in Case 1 ; and to Dr. J. W. D. Bull for many of the radiographs.

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CEREBRAL POLIOMYELITIS IN EARLY INFANCY

BY

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The cerebral type of poliomyelitis, although described by Parsons and Barling (1933), is now seldom reported. The occurrence of this variant of the disease in infants seems to be excessively rare. All forms of poliomyelitis are, indeed, uncommon in early infancy (Mouton, Smillie, and Bower, 1950). This paper describes three infants, all probably infected from a common source, in whom the disease was followed by severe cerebral symptoms.

Epidemiology

The outbreak occurred in the late summer of 1947, a season now notorious for the virulence and extent of its epidemic poliomyelitis. The *mise en scène* was a private nursing home in a small town in Berkshire. All three patients were born in this home, within two months of one another. At no time was there, however, any direct contact between the children or between their families. A midwife, Sister J., was in contact with the first affected child (Case 1) three days before the onset of his illness. She subsequently had close contact with the other two affected children. She herself had no illness suggestive of poliomyelitis nor had any others among the staff of the nursing home. After the third patient had developed his illness the home was closed. Table I shows the timing of the outbreak as a whole.

Case Reports

Case 1.—The mother had had one previous, normal pregnancy. Her pregnancy with the patient was uneventful, and she carried him to term. She was admitted to the nursing home on the morning of July 19, 1947, and half an hour later was delivered spontaneously of a normal male child. The labour was attended by Sister J. The child's birth weight was 7 lb. 10 oz.; he appeared quite healthy, cried at once, and sucked vigorously. He was given no injections of any kind.

The mother regained strength rapidly after delivery. She developed sore nipples on the tenth day and was advised to wean the child when she arrived home, which she did.

Mother and child left the nursing home, both in good health, on August 1, and went by car to stay at Henley in a large and isolated house, where there were three young children aged 5 to 10 years. Shortly after their arrival, on August 6, the owner of the next farm developed poliomyelitis and died within 24 hours: there was no known contact between the two houses at the time.

Mother and child returned home on August 26. Both were well, and neither of them went out, except in their car, during the next four weeks.

The midwife (Sister J.), who had delivered the child, visited him at home on September 3. She played with the child and fed him from a bottle.

On September 6 the child suddenly became restless and irritable. He cried continuously and appeared to be in some pain. He is thought to have been feverish. He remained in this state for 24 hours. On September 8 he took an hour to suck 4 oz. of milk from the bottle, but otherwise appeared to be normal.

The next day, September 9, he became very quiet and sleepy.

On September 10 the father developed a "cold" with malaise and fever, said to have been "just like a chill".

On September 11 the child's arms and legs were noticed to be limp and flaccid, he slept continually, was very slow with feeds, and seemed unable to cry. In the evening, in his bath, he suddenly gave a forced expiratory scream and went blue with lolling head. He recovered his normal colour and tone in a few seconds.

On September 13 he was seen by his family doctor. He was found now to be able to suck and cry normally: both legs and the right arm appeared to be wholly paralysed. The left arm was flaccid but some spontaneous movement was present. There were no other abnormal findings. The family doctor made a provisional diagnosis of poliomyelitis. On this day his elder brother developed a "chill" with malaise and headache.

He was seen in consultation (V.S.) on September 16 and the essential findings were as follows. The general condition was good: the cry was rather hoarse but strong. The lower left abdominal muscles were bulging and appeared paralysed but respiration was well maintained. The left arm appeared normal. The right arm was flaccid nor was there any movement at the shoulder. There was very slight flexion of the elbow and fingers, but none of the wrist, which was dropped. The right leg was completely flaccid, the only movement being an occasional twitching of the toes. There was slightly more tone in the left leg with a minimal contraction of the gastroc-

TABLE I

THE TIME OF THE DISEASE IN THE THREE CASES

Date 1947	Case 1 (E.B.)	Case 2	Case 3
July 19 ..	Born (**delivered by Sister J.**)		
Aug. 1 ..	Goes to Henley		
Aug. 6 ..	Neighbour died of bulbar poliomyelitis		
Aug. 26 ..	Leaves Henley		
Aug. 28 ..	7 days	Born (**attended by Sister J.**)	
Sept. 3 ..	**Sister J. calls, gives a bottle feed**		
	3 days	14 days	
Sept. 6 ..	Initial illness		
	5 days		
Sept. 11 ..	*Paralysis noticed. Convulsion* (Died 2½ years later)	*Leaves nursing home*	**Sister J.**
Sept. 16 ..		7 days	Born (**nursed by Sister J.**)
Sept. 17 ..		Initial illness	
		3 days	11 days
Sept. 20 ..		*Paralysis noticed*	
Sept. 25 ..			Initial illness
Oct. 5 ..			*Paralysis noticed.* (Alive but mentally deficient and paralysed.)
Nov. 4 ..		Died in convulsion	

nemius on pricking the foot. There was a little power of extension both of the ankle and toes. Tendon reflexes were absent in both legs.

Other systems appeared to be healthy.

A presumptive diagnosis of poliomyelitis was made and the family was advised on the precautions necessary for isolation. Arrangements were made for the admission of the child for orthopaedic treatment at the end of the infectious period.

During the next few days the child's general condition improved rapidly, and the left arm was used more freely.

He was admitted to the Wingfield-Morris Orthopaedic Hospital on September 26. Examination showed little change from the findings on September 16. The diagnosis of poliomyelitis was accepted.

A lumbar puncture on September 28 gave a clear fluid under normal tension which contained 50 mg. of protein per 100 ml.; no white cells were seen. The paralysed muscles remained unchanged during the next six weeks whilst under observation in hospital.

From this time, until the child's death, he was under regular supervision. No recovery of power in the legs took place. The right leg rapidly became flail except for a flicker of movement in the plantar flexors; subluxation of the right hip was found on January 21, 1949. In the left leg the flexors, abductors of the hip and

quadriceps were wholly paralysed, but a flicker of movement in the plantar flexors remained. The muscles below the right knee remained unchanged. Paralysis of the left lower abdominal muscles remained unchanged; the muscles on the right recovered some power during the six months after his illness. The right arm muscles recovered their power within eight weeks of the onset of the illness with the exception of the right deltoid which remained permanently weak.

In March, 1948, the child had a brief convulsion in which he threw up the left arm, turned the head and eyes to the left and appeared to lose consciousness. There were four similar attacks during the next two years. On April 10, 1950, he had a more severe seizure in which he suddenly lost consciousness, went blue, and vomited copiously; there was no twitching of the limbs. He recovered in two minutes, but, during the remainder of the day had four more small seizures.

Shortly before his death, in April, 1950, he was admitted to hospital for investigation.

Examination showed, in brief, a large, plump child with fair, curly hair and rounded features (Fig. 1). The child lay flat on his back, taking little notice of his surroundings, and making no sound except for an occasional grunt or roar. He made occasional aimless and ataxic movements of the arms, but did not attempt to play with toys. The grasp reflex was absent on both sides. Both legs were paralysed, the right being flail. Cranial nerves were normal, as were the fundi. Gross myopia was present. Examination of other systems showed no abnormality except for chronic infection and hypertrophy of the tonsils.

An electroencephalograph was made under seconal narcosis. A portion of the record is reproduced (Fig. 2), and it was reported as follows :—

> "When recording started, he was drowsy and soon fell asleep. Almost continuously there were paroxysmal, very high voltage spikes, and slow wave complexes focusing in the left pre-central area, but occurring in the right hemisphere also. These spikes became even more frequent as the 14 c./sec., 'light sleep rhythm', appeared.
>
> The record is grossly abnormal even for this age, and showed an almost continuous paroxysmal dysrhythmia, similar to a subclinical petit mal status, with an epileptogenic focus in the left pre-central region."

FIG. 1.—Case 1 shortly before death.

He was admitted to the Wingfield-Morris Orthopaedic Hospital on April 26, 1950, under Professor J. Trueta, for the manufacture of a plaster bed, which was applied without difficulty under seconal sedation that evening.

The next day he had some difficulty with breathing and there were bronchitic rhonchi in the chest. At noon he appears to have had a mild fit. During the remainder of the day he was restless with slow and shallow respirations and slight cyanosis. At 8.30 p.m. he suddenly stopped breathing and died.

Necropsy.—Necropsy was performed by Dr. Peter Daniel and Dr. Dawn Bosanquet 37 hours after death.

The child's body was well covered. Wasting of the lower limbs was not obvious as there was plenty of fat

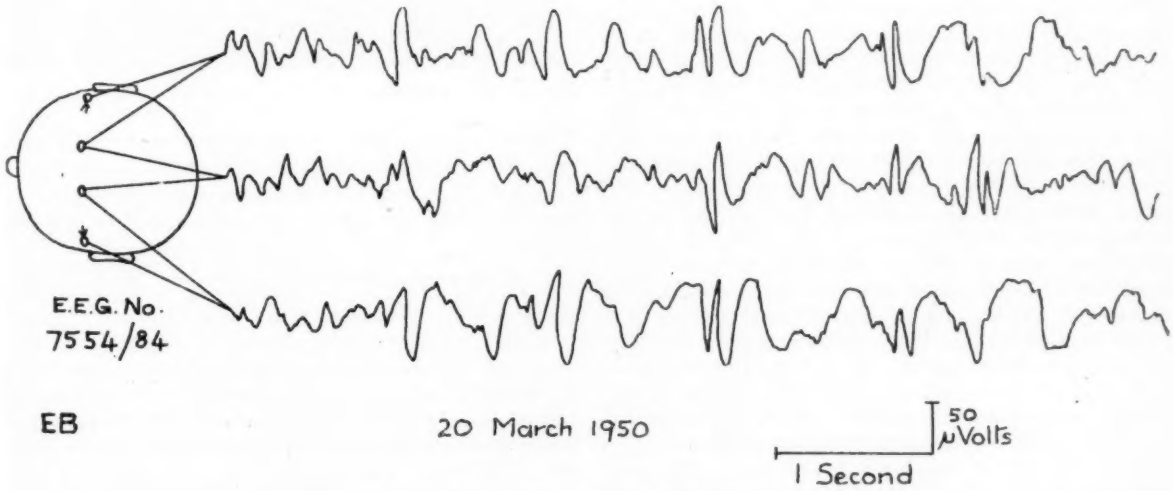

FIG. 2.—E.E.G. of Case 1 made shortly before death.

on them, but palpation showed that wasting of the muscles was, in fact, extensive. Both feet were inverted, the right more than the left. The head was large and the forehead slightly bossed, otherwise there were no visible deformities.

Thorax.—The trachea and main bronchi, in their lower parts, contained brown aspirated vomit. The upper lobes of the lungs were normal, the lower lobes contained aspirated vomit in the smaller bronchi and were congested.

Skull.—The fontanelles were closed but very marked " thumb-markings " of the posterior half of the vault were obvious.

Dura.—The dura was not tense and the venous sinuses were not thrombosed.

Brain.—The frontal regions showed some convolutional atrophy but the parietal regions appeared normal. There was a considerable increase in fluid in the frontal sulci. The base of the brain was normal and there were no internal herniae. The basal vessels were in good condition. Coronal sections through the cerebrum showed no macroscopic abnormality.

Muscles.—All the muscles of the lower limbs were grey, resembling fish muscle, except for the upper part of the hamstrings and adductors. There was an unusually thick layer of fat on the limbs which masked the shrunken muscles. The muscles themselves did not look fatty. The diaphragm, intercostal, and back muscles looked normal, as did the muscles of the neck, tongue, and eye. In the arms, the forearm muscles and biceps looked normal, but the triceps was grey.

There was marked fatty change in the liver and kidneys. There were no abnormalities in other systems.

The immediate cause of death appeared to be the inhalation of vomitus.

Histology.—The histological examination was performed by Dr. J. G. Greenfield, who reported as follows.

The spinal cord was examined at the levels of C.5, C.8, mid-thoracic, lower thoracic, lower lumbar, and sacral segments. At C.5 the number of anterior horn cells was greatly reduced on one side and considerably reduced on the other. At C.8 virtually all the cells had disappeared on one side and only a few of the ventrolateral group remained on the other. In the middle and lower thoracic regions there was little abnormality except for a fairly dense gliosis in the anterior horns, by comparison with the rest of the grey matter of the cord. A similar gliosis was seen in the more affected segments of the cord. In the lower lumbar segments no nerve cells were seen in the anterior horn of one side and only four or five on the other side. At the sacral level the population of the antero-lateral group was normal, but very few cells were seen in the anteromedial group. These lesions evidently corresponded to an old poliomyelitis but there was now no evidence of inflammation.

There were no abnormalities to be found in the brain stem, cerebellum, or basal ganglia. The cortex was well laminated in all areas. The motor cortex was not identified, probably because the brain had already been cut into slices when received. But it is possible that failure to recognize it was due to disappearance or atrophy of Betz cells. There were numerous nerve cells in the subcortical white matter, but they were normal at this age. No cause for the mental retardation was found.

A post-mortem radiograph of the skull cap (Fig. 3) confirmed a remarkable degree of craniotabes in the occipital region. This was thought to be due to the sustained pressure of the head, for during life the child lay immobile upon his back and was seldom moved from the supine position.

This child developed his disease at the age of 7 weeks. The initial illness and the subsequent

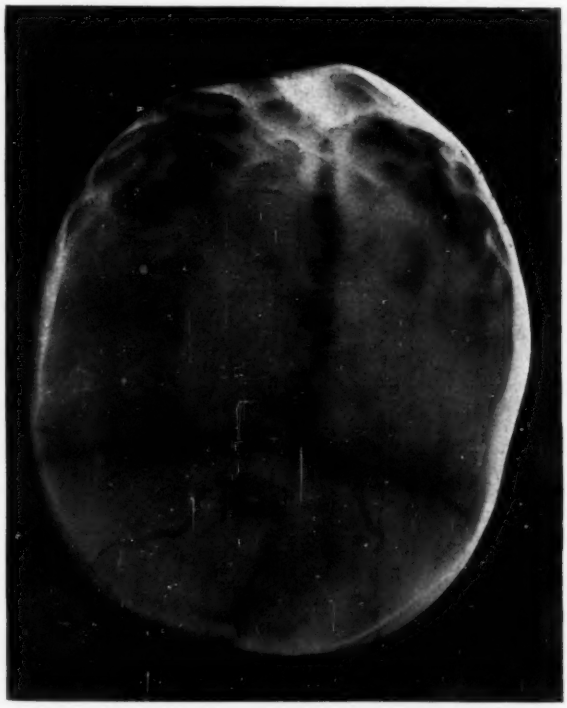

FIG. 3.—Radiograph (post-mortem) showing occipital craniotabes in E.B.

course of the lower motor neuron palsies were typical of spinal poliomyelitis. He later developed recurrent seizures with gross mental deficiency. At necropsy the changes in the spinal cord were those characteristic of an old poliomyelitis.

The other two patients are reported more briefly ; Case 2 was not seen by the authors.

Case 2.—R.A. was born in the same nursing home on August 28, 1947. The pregnancy and labour had been natural. The puerperium was without untoward events. The mother and child were attended by Sister J. There was a transient discharge from the child's nose and right eye at the age of 2 weeks.

At the age of 3 weeks he fell ill. He was feverish and appeared to be in pain, he cried continually and would

often draw up his knees as though taken with colic. A few days later it was noticed that the right arm was weak and flaccid.

On October 11, 1947, he was admitted to an isolation hospital. He was found to be a well developed child, and apart from his right arm, was without abnormal physical signs. The right arm showed a complete paralysis of the deltoid, biceps, and triceps ; movements of the wrist and hand were normal. A lumbar puncture made on October 28 showed a pleocytosis of 140 cells per ml. (26% polymorphs and 74% lymphocytes), but the fluid was heavily contaminated with blood.

The child continued in good health but without improvement in the paralysed muscles until November 3. On this day he had a convulsion with twitching of the face and deep cyanosis. He was put in an oxygen tent and the convulsion stopped. His temperature was found to be 102.8° F.

The next day, at 3.30, the child had another convulsion and, during the convulsion, died.

Necropsy was refused.

This patient was not seen by the authors and we are indebted to Dr. T. Fisher for details of the case. The history and clinical findings conform with the diagnosis of poliomyelitis.

Case 3.—G.H. (R.I.95400/49) was born on September 16, 1947. The pregnancy had been natural but labour was three weeks premature. The child weighed 5 lb. 12 oz. at birth, he appeared quite healthy, sucked vigorously, and moved all his limbs. Sister J. was in attendance.

The mother was a healthy woman with one elder healthy child. There was no history of familial diseases. All was going well when, on September 25, the child suddenly appeared lethargic and would not suck. The next day when seen in consultation he was ill with slight cyanosis, tachypnoea, and a temperature of 101° F. The whole body was seen to be oedematous. Crepitations were present at both bases. The skull and fontanelle were normal ; the neck and spine showed no stiffness. There were no paralyses. At the consultation it was decided to treat him as a case of bronchopneumonia and to give him a five-day course of intramuscular penicillin, and to feed him on expressed breast milk. He received 10,000 units of penicillin, intramuscularly, three-hourly. The injections were given into the thighs and deltoid muscles in rotation. On September 27 he was afebrile and appeared much better. On September 28 he had another peak of fever to 101° F. which lasted for 24 hours ; the generalized oedema was subsiding. Over the next few days the child's general condition rapidly improved, he regained vigour and sucked again with power. The left leg, however, lay flaccid, immobile, and oedematous.

Examination on October 7 showed that the left leg had in fact a complete flaccid paralysis of all muscle groups that could be tested. No other muscle groups were affected.

During the next few months the child progressed slowly and the left limb, in spite of a complete paralysis, grew normally in length. The child's general condition was good although it was noticed that he was tense and overactive.

During the latter part of his first year of life it was noticed that his hands were becoming useless. At the age of 8 months he had handled with ease a rusk, a rattle, and a doll : when he was 13 months old he was unable to grasp spontaneously and would drop a toy placed in his hands. At the same time spasticity began to appear in the right leg : this was first noticed at the age of 10 months as stiffness when the leg was handled. It was then seen that he did not move the leg spontaneously and that the toes were becoming clawed.

During this time he came under the care of Professor J. Trueta who observed that the child's mental development was grossly retarded. The child was on this account referred back to the Children's Department after an interval of one year and was admitted for investigation at the age of 16 months.

Examination on January 7, 1949, showed a retarded child unable to sit himself up. The tongue was constantly protruded, and there was a continuous fine flickering movement of the orbicularis oculorum. The arms were held up, with clenched fists, and were constantly moving, very like a pugilist shadow-boxing. The thumbs were adducted. Both arms were spastic with increased tendon reflexes. The right leg also was spastic and the joints appeared stiff and painful on passive movement. The left leg was flail. No tendon reflexes could be elicited in the legs. The abdominal reflexes were absent.

Other systems showed no abnormal signs. Radiographs of the chest and skull were normal. The cerebrospinal fluid was clear and the cytology and biochemistry within normal limits. The blood picture was within normal limits. An electroencephalogram was reported thus : "There was a little unstable alpha rhythm at 8 c./sec. on both sides. The record consisted mainly of runs of 5 to 6 c./sec. with occasional episodes of 3 c./sec. at medium voltage. These episodes started on the left side frontally, and then became bilaterally synchronous. The record was abnormal for this age, and the bi-frontal 3 c./sec. activity, starting on the left, may be a petit mal variant, although no typical wave-and-spike was seen."

Shortly after his admission he developed septicaemia due to *Bact. coli*. This illness ran a prolonged course, but was eventually controlled with sulphamezathine. He was taken home on February 13, 1949.

On April 17, 1950, he was re-examined. His mother thought that both his mental condition and his paralyses had remained unchanged during the past 14 months. He had had occasional "lapses", in which the flow of movement stopped suddenly, he stared, twitched his face, and then resumed his usual activity. These attacks had been variable : sometimes he had had several a day and at other times none for periods of several weeks. There had been no major seizures. His appetite was excessive and he appeared to be wholly impervious to cold. On examination, the condition of the child appeared to be static. His behaviour pattern was unchanged. He did not speak or show interest in his surroundings. He sat propped up making coarse

athetoid movements of the arms. His grimaced constantly and gibbered vigorously. When given a spoon he perseverated on a simple banging movement against the side of his pram. The left leg was unchanged, remaining flail, wasted, and immobile. There was, however, no shortening compared with the right leg. The arms and right leg showed no change from their condition a year before.

Other systems showed no abnormal signs.

This child had what appeared to be a simple attack of spinal poliomyelitis shortly after birth involving the left leg. He later developed spastic paralysis of the remaining leg and both arms with gross hyperkinetic mental deficiency, athetosis, and recurrent minor epileptiform seizures.

Discussion

Epidemiology.—The first six months of life are generally considered to be a period of marked immunity to poliomyelitis. Between 1897 and 1948 only 17 such cases appear in the literature. It is remarkable, therefore, that Mouton and others, (1950) were able to find 10 cases in a single epidemic. Many other reports of single cases have in fact been published recently (Garlick, 1948 ; Brandt, 1946 ; Biermann and Piszczek, 1944 ; Shelokov and Weinstein, 1951). The disease in several of the cases derived from the mother. Shelokov and Weinstein suggest that in their case the transmission of the virus occurred *in utero*. The majority of women affected with poliomyelitis at term, however, give birth to healthy children.

We have suggested that the midwife, who attended the three children described in this paper, was the vector. She was entirely healthy throughout the whole episode. Adamson, Moody, Peart, Smillie, Wilt, and Wood (1949), in their elegant study of the ecology of poliomyelitis among the Arctic Eskimo, have shown that the clinically healthy human carrier is the chief vector of the disease. Sweetnam (1948) has given evidence of this mode of spread in England ; Hahn and Taylor (1947) arrived at similar conclusions for a German epidemic, as did Nissen (1947) for the St. Helena outbreak. In our cases the midwife was the only person to have contact with all the affected children. It appears probable that the virus entered the family of E.B. (Case 1) at Henley and was manifested in E.B. as florid poliomyelitis and in his father and brother as an abortive febrile illness. The midwife may have acquired the virus at her visit on September 3 (Table 1), and may later have transferred the disease to the other two children. The evidence is necessarily wholly circumstantial. If indeed this was the route of the disease the incubation periods were of the order : Case 1, 10–30 days ; Case 2, 7–14 days ; Case 3, 9 days or less.

Clinical Features.—The syndrome of " cerebral " poliomyelitis finds little place in the recent literature, although it is mentioned by Wickman ; and the syndrome, as it appeared in our patients, was described in full by Parsons and Bareing (1933).

Bodian (1947), in his classical study, showed that widespread encephalitic foci were invariably found in patients who died in the acute phase of the disease, and he remarked that, to the pathologist, both experimental and human poliomyelitis were invariably encephalitic. Clinical signs of cortical dysfunction were shown by Brown, Baker, Adams, and McQuarrie (1947) to be common in the acute phase of the bulbar variant. They found that these symptoms resolved when oxygen was given and concluded that they arose largely from cerebral anoxia. Crothers and Meyer (1946), in a psychological assessment of 15 children who had had encephalitic signs in the course of poliomyelitis, found in all cases some minor mental disturbance, usually taking the form of visuomotor deviations.

There appears to be little doubt that the poliomyelitis viruses may cause both pathological and clinical lesions at any level of the cerebrospinal axis. Clinically manifest lesions above the level of the cord are, however, transient in the vast majority of patients.

The three children we have described presented in the initial stages with acute biphasic febrile illnesses, followed by multiple lower motor neuron palsies, and appeared to have the simple spinal type of the disease. This was followed, however, in the two survivors by the slow unfolding of the signs of mental retardation with epileptic seizures. Brewis and Neubauer (1948) have described a girl of 17½ who, in the course of polioencephalomyelitis had convulsions, but this complication is excessively rare. Mouton and others (1950) found no case of convulsions during the acute phase of poliomyelitis in infancy either in their own series or in the literature ; they did not, however, present the results of a follow-up study of their cases. Mental deficiency is notoriously elusive in the first year of life, whilst epilepsy commonly matures silently between a primary insult and the onset of seizures. Both these features were apparent in our patients. Convulsions occurred in all three and in two cases were directly associated with the death of the children. In Case 2 there was a lapse of 46 days between the onset of the poliomyelitis and the child's death during a convulsion. In Case 1 death followed some hours after a seizure, and seemed to have been due to the inhalation of vomit while the child was unconscious. This child's epilepsy appeared seven months after the acute phase of the disease. In Case 3 minor epileptiform seizures appeared in the third year of

life. The lesions underlying the gross mental deficiency of these children were not defined, and at necropsy the brain of E.B. showed neither gross nor microscopic evidence of cerebral destruction on sectioning. On the other hand, the electroencephalographic records showed severe abnormalities, and presented in Case 1 a pattern resembling the status of petit mal. It is interesting to speculate upon the role of the chronic electrical dysharmony as a causal factor in the dementia of these children.

One final point should be mentioned. The diagnosis of poliomyelitis in the preparalytic phase is a matter of extreme difficulty in early infancy. The ordinary physical signs of meningeal irritation are often absent in affected infants, (Mouton and others, 1950) and these signs were absent in the children in the present study. Similarly, neck rigidity is usually absent in neonatal meningitis (Jackson, 1950). Unexplained fever with lethargy and pain on fondling, or enlargement of the skull, demand diagnostic lumbar puncture at this age.

Summary

A small outbreak of poliomyelitis in a nursing home is described.

The patients were infants of 7, 3, and 2 weeks of age. All had multiple lower motor neuron palsies. One died in convulsion during the convalescent phase of the disease. One child survived for two years and nine months but was affected with multiple palsies, idiocy, and fits. He died after a seizure and showed at necropsy the typical lesions of poliomyelitis in the spinal cord. One child remains alive but is also an epileptic idiot with upper and lower motor neuron palsies and athetosis.

The epidemiological and clinical aspects of the syndrome are discussed.

We wish to express our gratitude to Dr. J. G. Greenfield for making a histological study of Case 1, and to Drs. Peter Daniel and Dawn Bosanquet for the necropsy report. We are indebted to Dr. T. Fisher for the details of Case 2, and to Professor J. Trueta for permission to quote his observations on Cases 1 and 3.

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NEUROPATHIES CAUSED BY BONY SPURS IN THE CERVICAL SPINE WITH SPECIAL REFERENCE TO SURGICAL TREATMENT

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Osteophyte formation, in association with prolapsed cervical disc, has previously been reported. The disc was thought to be the only important factor from the clinical point of view until Bull (1948) studied the radiological evidence and measured the size of nuclear material at various cervical levels. He doubted the prime role of the displaced disc in the production of neurological symptoms and stressed the arthritic changes in the neurocentral joints. Philip (1950) further emphasized the importance of osteophytes in syndromes of root pain in the upper extremities.

The difficulties and dangers attendant on the surgical approach to the cervical encroachment syndrome have previously prevented full recognition of the deformities responsible for the neurological syndromes found at these levels. This paper is based upon operative, radiological, and clinical observations, and will emphasize the importance of bony spurs compared with the disc prolapse. A more exact scrutiny of the deformity and its anatomical and mechanical relationship to nervous structures has resulted from improvement in surgical technique. Numerous cases of cervical root and cord disturbances have been studied clinically and radiologically. Of these, 19 failed on conservative treatment and operation was performed to prevent progression of symptoms. These patients showed intractable brachialgia or progressive root and cord dysfunction, or both together. A minute portion of prolapsed disc tissue was found in only one of 19 cases, while in all cases there was an osseous spur at one or more levels, with varying degrees of hypertrophy of the overlying posterior common ligament. In all cases the spur formed a bridge, traversing the thecal canal from one intervertebral foramen to the opposite and the adjacent margins of each body contributed in the formation of the spur. The morphological features of the bridge and its anatomical relationship to the multiple nervous structures crossing it will be described later. Such spurs are so placed that they may interfere with many neural elements. Eight cases are reported, illustrating the more common syndromes and the possible resemblance to other conditions, such as progressive muscular atrophy or subacute combined degeneration of the cord. They demonstrate too the variability of cervical root disorders, occurring alone, or with cord involvement. Either nerve root or spinal cord signs may appear first and either may remain predominant throughout. With regard to brachialgia, operation was undertaken when root pain resisted all forms of conservative treatment, or when it merely represented one part of a wider syndrome with cord involvement. In all 19 cases the bony bridge was removed in its major part, or in its entirety. Post-operative follow-up is recorded for periods of up to 27 months. A posterior laminectomy alone was carried out in one case (No. 8) at the level of the spur, which at this time was thought to represent a venous malformation. Temporary improvement resulted, followed by further deterioration. Case 7 demonstrates that spurs in the low dorsal and cervical regions can produce two separate disabilities.

Case Reports

Case 1 (No. 3797).—A man aged 38 years, a salesman, on April 6, 1949, complained of pain in the right arm since 1942, and of weakness and wasting of this limb, with wrist drop since 1945. He was involved in bomb blast in 1942, and dates the onset of pain in the arm from this time. He was then in the Forces. He developed a schizophrenic psychosis and was given full insulin therapy, which aggravated the neuritic symptoms. There was a good recovery from the psychiatric condition, but suddenly in 1945 he found that the right hand was weak in its grip and clumsy in handling small objects. Later he could not write and noted fibrillation in small muscles of the hand. He was seen at the National Hospital for Nervous Disorders, Queen Square, London, and was examined by three consultants. Extracts from their reports read :

". . . I can but agree with the diagnosis of progressive muscular atrophy (made first in 1945 on muscular weakness, wasting and fibrillation of arm muscles), and I think he is, in addition, developing a certain amount of spasticity as shown by increased reflexes. The prognosis is, of course, extremely bad and I have been disappointed with all forms of therapy advocated in this disease."

". . . It has been suggested that his symptoms are due to herniation of an intervertebral disc, but I could not discover clinical or *x*-ray evidence of this. The minimal sensory symptoms are also against this diagnosis. My own impression was that he has an anomalous form of progressive muscular atrophy and Dr. . . . came independently to the same diagnosis. I must admit that I am not wholly satisfied with this diagnosis, but after careful investigation of his case I could come to no more probable conclusion ".

He consulted us on April 6, 1949. Pain was now down the ulnar side of the left arm and over the lower right ribs at the back. The neck movements were full, but extension produced pain in the right side of the neck, radiating to the shoulder tip.

Cranial nerves were normal.

Motor Functions.—There was wasting and weakness in the right pectoralis major, supra and infra-spinatus. The shoulder girdle drooped slightly on the right. Weakness and wasting were well marked in the triceps, the forearm, and hand muscles on the right, and most marked in the extensors of the wrist and fingers, the thenar, and hypothenar muscles. It was less marked in the biceps, deltoid, and spinati. The right scapula was winged. Fasciculation was continuous in all the affected muscles, and muscle tone was reduced on the right. In the left upper limb there was subjective weakness of the hand, but little objective cause could be found. Sweating was profuse over the ulnar borders of both hands.

In the lower extremities power and tone were normal and symmetrical.

	Right	Left
Deep reflexes		
Biceps	++	+
Triceps	—	±
Supinator	±	+
Knee	++	++
Ankle	+	+
Superficial reflexes		
Abdominal	+	+
Plantars	flexor	flexor

With these should be compared the findings in 1947 at the National Hospital that " all the reflexes in his right arm are absent and those in the left depressed ".

Sensation.—Pain, touch, vibration, and joint sense were normal throughout. A radiograph showed narrowing of C.5-6 and less of C.6–7 interspaces. In the right oblique there was narrowing of the nerve canal at C.5–6.

Operation.—On April 20, 1949, under pentothal, gas, and oxygen, a midline neck incision was made and laminae C.5 and 6 were removed. A bony deformity, anterior to the cord and producing kinking and posterior displacement, was found. There was marked constriction of the mixed roots C.5 and 6 on the right. The deformity was seen to be due to an osteophyte formation anterior to the roots at both levels. Anterior and posterior decompression was carried out on the right and this freed the two mixed roots. On the left only a posterior decompression was done. On this side the bony spurs, although present, did not reach the same height. On both sides the dentate ligaments were cut and the dura was closed. At operation the cord was seen to be compressed and immobile.

No new signs were detected in the immediate post-operative period. Twenty-four hours later the patient volunteered that his fingers were " freed ". Six days later there was subjective improvement in abduction and adduction of the right fingers and extension of the right thumb. On discharge on May 18, 1949, improvement was still mainly subjective.

Six months later he reported a gradual improvement in " freeness and power " of all movements of the right arm and hand. Wasting and fasciculation were still present in this hand, but thenar and hypothenar muscles were fuller and power had improved. Pronation was still very weak. Extensors of the wrist, pectoral muscles, biceps, triceps, deltoid, and supraspinatus were almost normal. In both arms the reflexes were all present and slightly increased. In the left arm power, tone, and facility of movement were normal.

The lower limbs were normal and no abnormality of sensation was discerned.

This man's condition had been diagnosed by three eminent neurologists as progressive muscular atrophy. There is, therefore, no need to stress the similarity of the syndrome to this condition, but it might be noted that pain was perhaps too prominent. The fluctuation in the reflex findings over four years and the complete absence of sensory abnormality despite involvement of the mixed nerve roots is of interest.

Case 2 (No. 4335).—A man aged 73 years, an ex-railway guard, was examined on August 20, 1949, and complained of loss of normal movement and sensation of the hands and legs.

Ten years previously he had noticed " numbness " of the legs, but this disappeared after two months. Seven months before examination it recurred, mainly in the feet, to be followed by weakness of the right, then the left leg. Weakness gradually progressed and he fell on three occasions. Frequently there was a feeling of " muslin cloth " wrapped around his feet. Then the arms became " numb ". He became unable to write as he could not hold a pen properly, and dropped objects without noticing. The hands were shaky and he was unable to play the piano. Distinction of heat and cold in the arms and legs deteriorated. The right arm was weaker than the left. Shooting pains in the neck and

shoulders were worse on the right. Micturition was normal.

There was slight pallor of the optic discs, but cranial nerves were otherwise normal.

Motor Function.—Power was reduced in both arms and legs, more so on the right. He was unable to stand for long. There was weakness of all muscle groups in the lower limbs, with increased tone and clonus, especially on the right. In the upper extremities power was generally good, but there was slight weakness of finer finger movements, with commencing wasting of the first dorsal interosseous on the right. There was some subjective difficulty in using the hands.

Sensation.—Vibration and deep sensibility were markedly impaired in all extremities. Pain, touch, and temperature appreciation was grossly depressed in both legs. There was a variable sensory level about D.11–12. While under investigation pain loss appeared in C.6 and 7 on the right, and a sensory level became stabilized on the trunk at D.3.

	Right	Left
Deep reflexes		
Jaw	+	+
Triceps	++	++
Biceps	++	++
Supinator	++	++
Knee	+++	+++
Ankle	+++	+++
Superficial reflexes		
Abdominal	—	—
Plantars	Extensor	Extensor

Blood pressure was 165/85, and pulse rate 80 per minute. The heart borders were normal to percussion and there were no murmurs, but many extrasystoles. A blood count showed mild anaemia, but no abnormality in the red cells was observed.

A test meal (June 28, 1949) showed achlorhydria.

There was a slow rise and inadequate fall of the cerebrospinal fluid on jugular compression (1 lymphocyte per c.mm.; total protein, 63 mg. per 100 ml.).

Radiographs showed marked generalized osteoarthritic lipping throughout the vertebral column. "Encroachment" was demonstrated into the foramina on each side at C.6–7 and C.7-D.1.

Lipiodol myelography showed a temporary "flat type of cupola" formed opposite interspace C.7–D.1 and C.6–7 in the prone position. Lateral views showed filling defects at these interspaces.

Operation.—We operated on September 13, 1949, using pentothal and gas and oxygen anaesthesia.

With a midline incision a laminectomy of C.7, the lower portion C.6, and the upper portion D.1 was performed. The dura was opened. An obvious spur, bridging from side to side, and formed by contiguous borders of C.6 and 7, was found. This involved the mixed roots at C.7 and the anterior radicles of C.8, which were already discrete at C.7 level and placed more medially. On flexing the neck there was posterior displacement of the cord, producing pallor. Posterior and anterior decompression was performed to free nervous structures.

A smaller spur was found at C.7–D.1 with involvement of mixed roots of C.8 and anterior radicles of D.1. On flexion of the neck, pallor of the cord was less marked at this level. Only posterior decompression was carried out. Dentate ligaments were cut on both sides over these segments.

Owing to the age of the patient anterior decompression of the roots and cord was less extensive than in Case 1.

There was no immediate accession of neurological signs, and10 days later the patient felt the numbness fast disappearing from the legs and the pains from the arms. He was discharged on November 11, 1949. Superficial healing of the wound was temporarily delayed.

He was last seen in September, 1951, when he was walking unaided. He carried a stick for confidence, although this was not necessary. The legs were still tiring after much exercise. Movements of the neck were absolutely free. He was now able to play the piano with adequate dexterity.

Examination demonstrated no impairment of the long tracts. The root signs were not elicited. In the lower limbs there was depression of vibration sense only, compatible with age. There were no other sensory signs. Blood counts and test meal were not repeated.

Subacute combined degeneration of the cord was mooted at one time. The cord alone was first involved and later the roots. Only at a late stage could a clinical neurological level be determined.

Case 3 (No. 5017).—A man aged 36 years, a parks superintendent, was examined on March 9, 1950. He complained of pain down the left arm and from the shoulder referred to the radial side of the forearm and index finger. Latterly he had perceived numbness in the index finger. In 1944 he was thrown out of an army lorry, and there was some indefinite injury to the neck. Pain appeared about two months later coincident with some domestic troubles. He had previously been seen by neurologists, who diagnosed a prolapsed cervical disc. Physiotherapy, neck traction, and later collar immobilization all failed to give relief.

On examination no motor involvement was detected, but there was alteration in the quality of sensation in dermatomes C.6 and 7 on the left, and to a less extent in C.5. There was no evidence of cord dysfunction. The left biceps reflex was depressed.

Radiographs showed a massive osteophyte formation, formed by the posterior lips of the bodies of C.5 and 6. The osteophyte was very evident in the left intervertebral foramen and extended across anterior to the cord, this being well shown on the lateral plates. There was narrowing of the interspace with a cervical kyphosis.

Operation.—On March 9, 1950, under general anaesthesia a window of bone was removed, leaving virtually intact the laminae and spines at C.5–6 Mixed nerve C.6 was exposed and this was very flattened over an anterior spur, the "high-spot" of which was just medial to the axilla of the nerve. The spur extended laterally

into the foramen. The spur was removed anteriorly over its whole visible and palpable extent.

By the second day the arm pain disappeared. Between the sixth and ninth day it reappeared slightly and then again disappeared. The patient was discharged on March 28. There was still some neck stiffness, but no abnormal signs. He had no other subjective complaints. He was last seen in August, 1951. There was still some stiffness of the neck, rather less than before. Passive movements of the neck were free in all directions. There was occasionally a transient twinge of the former pain in the arm. He had returned to work which involved lifting and moving heavy objects. Radiologically the cervical kyphosis persisted.

This patient illustrates the group in which brachialgia is the presenting and dominant symptom. Again despite involvement of a mixed nerve root there was no paresis or marked objective sensory loss.

Case 4 (No. 4513).—A man aged 47 years, a business man, was examined on January 1, 1950. He complained of progressive weakness in the right leg for one year. He had suffered from flat feet for many years, and thought that this caused pain in the calves. One year before the right leg began to drag, and had become considerably weaker than the left leg. He had no pain or discomfort in the limb. He had been seen by many orthopaedic surgeons and had been given various forms of physiotherapy. The picture was one of slow deterioration, no injury, and never any lumbago or sciatica. He had no bladder or bowel symptoms.

On examination it was seen that he walked with a slight limp. The cranial nerves were normal, as were the upper extremities.

There was slight wasting of the right quadriceps, and marked reduction in power of all movements of the right lower limb, particularly, however, in the flexors of the thigh and dorsiflexors of the foot. Within limits of power there was no incoordination and no fasciculation.

	Right	Left
Deep reflexes		
Biceps	++	++
Triceps	++	++
Supinator	++	++
Knee	+++	++
Ankle	+++	++
Superficial reflexes		
Abdominal	+	+
Plantars	equivocal	flexor

In hospital both plantars became extensor in type.

Myelography demonstrated an indefinite defect at D.12. Here also was a doubtful pedicle widening. In the prone position throughout the whole cervical region there was a "stickiness" in flow.

First Operation.—On February 2, 1950, under general anaesthesia, and acting on the minimal radiological evidence, a negative exploration was performed at D.11–12. No change in his condition resulted.

In subsequent months there was a deterioration of power in the upper and lower limbs, particularly the right. The lower limbs became so weak that the patient was unable to walk. Apart from subjective numbness in the third and fourth fingers of the right hand there was no sensory disturbance. Intermittent fasciculation was noticed in the muscles of the upper limb. There was no sphincter disturbance. Objectively, weakness of all limbs was confirmed. The deltoid, extensors of the fingers, and the small muscles of the hand, particularly those of the thumb, were weak and wasted. There was no further wasting in the legs, but spasticity was present.

All deep reflexes were increased. Both plantars were persistently extensor. The jaw and pectoral reflexes were increased. A slight loss to vibration was found over the malleoli.

Radiographs showed extensive spur formation, especially at C.5–6, 6–7, and C.7–D.1, and less at C.4–5. C.5–6 interspace was narrowed.

Second Operation.—On May 1, 1951, the laminae of C. 5, 6, and 7 were removed. Intrathecal inspection showed marked pallor and immobility of the cord. Posterior displacement of the cord coincided with the anteriorly placed spurs at C.5–6, 6–7, and C.7–D.1. These spurs were entirely removed and the cord was replaced in a more natural position with resulting increased mobility.

After operation there was no accession of neurological signs, and after 48 hours there was subjective freedom of movement of the ankles and feet, and clonus was less. After three days the right plantar response was flexor. Thereafter the right plantar reflex was variable and bilateral clonus was occasionally found.

At the fourth week he started walking and could take an average good step.

After six months he reported that his hands were more powerful and he was able to use them for everyday actions. Wasting was now less obvious and deep reflexes of the arm were normal. Clonus in the legs was ill-sustained and the plantar reflexes were variable. Psychologically one felt that this man was somewhat frightened of himself and was not exerting the full available power in his legs.

This case is reported mainly to draw the moral from the rapid progress between the two operations. The increase in disability was largely motor and referable to the lower limbs. Only at a late date did clinical signs appear to suggest a cervical level. The follow-up is too short to assess the effect of the second operation.

Case 5 (No. 5143).—A man aged 45 years, a surgeon, on May 7, 1950, complained of progressive weakness of the left hand for 15 months.

Six months before the onset of disability he fell from a chair and hit his head against a wall. Some stiffness of the neck followed and he then noticed weakness of the small muscles of the left hand, at first when closing artery forceps. Weakness progressed and wasting became so extreme that he became unable to operate. There was no pain, no undue sweating, and no fasciculation.

On examination there was no Horner's syndrome and the cranial nerves were normal.

In the left upper limb below the elbow the musculature was reduced in bulk. There was marked wasting in the muscles of the thenar and hypothenar eminences, while the interosseous muscles and lumbricals were severely affected with a corresponding loss of power. Weakness of flexors and extensors of the wrist and fingers was however minimal, despite the wasting of these muscle groups. The other limbs were normal.

The deep reflexes were symmetrical in both upper and lower limbs. Plantars were flexor.

Sensation.—This was normal to all modalities throughout.

Radiographs showed C.6–7 interspace narrowed, and spur formation on lateral and oblique views at C.4–5 and at C.6–7.

Operation.—On May 25, 1950, under general anaesthesia, " windows " were made at C.6–7 and C.7–D.1. A spur at C.4-5 was ignored as incapable of producing the clinical picture. Clinically the level C.7–D.1 was more implicated, and despite negative *x*-ray findings a window was made here. A large spur was found at C.6–7 and was removed anteriorly. There was compression of the mixed root of C.7 and anterior root of C.8 at this site. Nothing abnormal was found at C.7–D.1.

In this case the spur had its " high-spot " beneath the upper axilla of the C.7 root. There was relative post-fixation of the emergent root. The height (or depth) of the spur reached $\frac{3}{16}$ in.

There was no immediate deterioration. The patient was seen in January, 1951, seven months after operation. He had returned to surgery, but required trick movements to handle instruments. However, before operation he was quite unable to operate. The bulk of the first dorsal interosseous muscle had decreased further but in all other movements power was improving. Particularly it was to be noted that there had been no extension of the muscle groups affected, nor had there been any increase in the weakness of the affected muscles. Before operation this surgeon had been given faradism, shoulder girdle exercises, and a course of " stimulating " *x*-ray therapy to the cervical spine. In spite of these measures his condition had progressively deteriorated.

In this case there were no sensory abnormalities, nor was there a history of pain. The case again demonstrates the dissociation of extreme, focal motor loss without sensory disturbance, although mixed cervical roots are involved. The progression of wasting in some of the intrinsic hand muscles without spread to other groups might indicate that the affection of C.8 motor root was too complete to be relieved by operation.

Case 6 (No. 5301).—A man aged 44 years, a farmer, consulted me on July 20, 1950.

In January, 1950, driving on a long journey, he developed pain at the back of the neck, radiating to the left shoulder. Two days later the neck became stiff. He had physiotherapy and *x*-radiation without improvement. In March, 1950, the right side of the trunk and right leg became numb. Numbness in the leg was more marked below the knee. At the same time the leg became weaker and dragged when he walked. There was, however, no limp. In June, 1950, numbness of the ulnar side of the left palm and to a lesser extent of the right palm was noticed. In July, 1950, the left hand-grip became weak and again to a lesser extent the right grip. All objects touched with the palms were cold. He had precipitancy in the mornings, but no other disturbance of micturition. When the bladder was full, sensation was appreciated only on the left side of the lower abdomen. Sexual functions were normal. He was sleeping badly. Two weeks before examination there was a sudden deterioration of power in the left hand, but there had been no further deterioration since then. Pain in the back of the neck was still present, but had been much less severe for one month. It was aggravated if he leaned forward in a sitting position.

On examination the patient was seen to be obese, with no limp. There was a large port-wine stain over the left trunk and left buttock and upper thigh. This did not cross the midline and had been present since birth. The neck movements were normal in range, but extreme extension produced a pain, radiating to the left shoulder. This pain was similar in nature to that which marked the origin of his illness. The cranial nerves were normal.

Motor Functions.—Power was reduced in the left arm. There was general weakness in all muscle groups, but most marked in the small muscles of the hand. The right arm was normal, tone was symmetrical, and posture was well maintained. There was some clumsiness of fine and alternating finger movement on the left.

There was a general reduction of power in the left lower limb, but tone was normal.

	Right	Left
Deep reflexes		
Biceps	+	++
Triceps	++	+++
Supinator	+	++
Knee	++	+++ (clonus)
Ankle	+	+
Superficial reflexes		
Abdominal	—	—
Plantars	flexor	flexor

Sensation.—Pain, touch, and temperature sense were depressed in C.8 and D.1 on both sides. On the left C.7 was slightly involved to the same modalities. Vibration was slightly reduced in the same distribution on the left. Joint sense was intact.

Pain, touch, and temperature sense were less well appreciated on the right side of the trunk below D.3. The depression extended to the midline.

Pain, touch, and temperature sense were depressed in the right lower limb. Joint sense was intact and vibration sense was absent.

General examination was normal.

Pre-operatively this was thought to be an angiomatous malformation of the cord, with a solid tumour at about C.6–7. Myelography was performed and showed "stickiness" of flow upwards from D.12 with a "flat type" of cupola at C.6–7 interspace. The block was, however, partial.

Operation.—On August 10, 1950, under general anaesthesia laminectomy at C.5 and 6 was performed. The dura was opened and the cord was found to be kinked at two levels, opposite C.5–6 and 6–7 interspaces, displaced posteriorly, flattened from side to side, and pale at the operation site. At each of these levels there was spur formation. The upper spur involved the mixed root of C.6 on each side and at this level the anterior roots of C.7 were implicated in front of the cord. Decompression at this level was performed anteriorly from side to side. The spur between C.6–7 did not extend laterally. Its "high-spot" was in the midline. It involved the cord and the anterior radicles of C.8. The position of this spur precluded its removal with safety at this sitting, as much handling had already occurred at the higher level. It was left, and a posterior decompression with section of the dentate ligaments performed at this zone. The posterior ligament was thickened to $\frac{1}{8}$ in. over the upper spur. An angiomatous malformation of minor degree was found over the length of the exposed cord, but considered insufficient to cause the major symptoms.

On recovery from the anaesthetic the patient moved all limbs and stated that the numbness of the right leg had disappeared. He passed urine normally. He was walking on the fifteenth day, and discharged on September 6, just under one month from operation. He returned to work after two months.

One year later numbness in the trunk and leg had disappeared, apart from a small zone on the outer side of the right calf. There was no weakness of the upper or lower limbs, or pain or stiffness of the neck. He still felt numbness in the ulnar borders of both hands.

All reflexes were normal. There was some diminution of pain in C.8 distribution on both sides, but no loss elsewhere. Bladder sensation and function were normal.

This patient finds the numbness in C.8 distribution an annoying disability. He is very willing for a second operation if this could be eliminated. It is possible that the C.6–7 spur anterior to the cord may yet have to be dealt with, especially as more serious symptoms may eventuate.

It is noted in this case that there was an abnormality of vascular supply to the spinal cord, presupposed at clinical examination and confirmed by inspection at operation; yet evidence is here adduced to show that the congenital abnormality had but little influence in the aetiology of the syndrome presented. However, one cannot but speculate on the role which this abnormality subserves, when compression of the cord is produced by bony deformity or by any other means.

In this case spur deformity was found at operation and the radiological diagnosis made only in retrospect. The case indicates the ability of a bony spur to produce a predominantly sensory defect in terms of the cord conduction tracts. Only at a late stage did sensory, motor, and reflex signs, referable to root involvement, appear.

Case 7 (No. 4573).—A middle-aged woman consulted me on January 2, 1950, and complained of pain in the back and lower limbs. She had had a serious fall four years previously, with no sequelae at the time. In June, 1949, while bending to arrange papers her back became "locked" and she was unable to straighten up. A few hours later, on attempted movement, there was pain low in the back and down the right thigh. This continued and grew more severe. There was temporary relief on resting. Pain recurred in August, 1949. Conservative treatment and manipulation under anaesthesia gave no relief. She became incapable of any occupation requiring much movement. The left leg became weak with dragging of the foot.

On examination the upper limbs, trunk, and cranial nerves appeared normal. There was considerable weakness, with wasting of the glutei, hamstrings, quadriceps and peronaei, more on the left, but no fasciculation. Tone was normal. The left lower abdominal reflex was absent and the deep reflexes were depressed in the left leg. Sensory loss was apparent to all modalities to the level of D.12–L.1 on the left, most marked to pin prick. Vibration was lost to the iliac crest, but joint sense in the left great toe was good. Movements of the back and left leg were restricted by pain. There was considerable spasm of the paravertebral muscles.

Myelography showed a filling defect at the junction of D.9 and 10, outlining a medial lesion anterior to the cord.

Operation.—On January 12, 1950, a laminectomy at T.9 and 10 was performed, and a small, mushroom-shaped, bony spur was removed from the midline, anterior to the cord. In the words of the surgeon: "it hardly seemed as if it would account for her symptoms", but the thecal canal was narrow at this point.

Within one week there was disappearance of the previous pain, and improvement in sensory loss. She was discharged on February 2, 1950, with residual stiffness of the low back related to the operation wound; there was no sensory residue, or dragging of the left foot.

This case has been introduced to indicate the role that bony spur formation may play in producing progressive cord or root dysfunction at levels other than the cervical spine—here in the low dorsal region—and also in my experience at the lumbosacral region, where it may bear partial responsibility for the syndrome of a prolapsed disc.

Case 8 (No. 4424).—A housewife aged 47 years was seen on October 22, 1949. In December, 1945, she developed pain in the right arm and shoulder, radiating into all the fingers. Before this she had been nursing her father in war-time Holland, under difficult conditions

and at times carried him on her shoulders. The onset was sudden with a sense of uselessness of the hand. She was unable to write easily and dropped things. She experienced "pins and needles" and "shocks" of indescribable pain in the hand and forearm. At the end of 1946 the right leg became weak and dragged. Treatment with physiotherapy, infiltration of "fibrositic" nodules, and neck-traction was given for five weeks between January and March, 1949. Paraesthesia improved initially, but afterwards there was aggravation of pain. Thereafter she reported steady deterioration; there was also occasional pain in the left arm and attacks of clockwise vertigo.

On examination there was no nystagmus, and no evidence of vestibular or cerebellar dysfunction. Neck movements were limited, especially extension. There was weakness of the intrinsic muscles of the right hand, flexion of the wrist and fingers, pronation and supination of the forearm, and slight weakness of extension of the wrist and elbow and of the deltoid. There was wasting of the first interosseous muscle on the right. Biceps, supinator, and triceps jerks were depressed on the right. Vibration and joint sense was depressed to the shoulder, and pin prick was depressed in C.6 and 7 dermatomes on the right. In the right leg there was weakness of plantar and dorsiflexion, with increased tone of the distal muscles. There was an increase of right knee and ankle reflexes with clonus and extensor plantar responses. Abdominal reflexes were all absent. Blood and cerebrospinal fluid were normal. Manometry gave normal results.

Radiologically there was loss of the disc space with spur formation at C.4–5 and C.5–6. Spur encroachment was clearly evident in the right and left oblique views at the lower level. The whole body of C.5 appeared to be drawn backwards breaking the contour of the spinal canal at both levels.

At this time it was felt that the bony deformity was incapable of producing such a disability unless a prolapse of the disc was also present.

First Operation.—On November 3, 1949, through a midline incision laminectomy at C.3, 4, and 5 was performed. The theca was opened. The cord was displaced slightly backwards. On rotation a bulge was felt and seen anteriorly and extrathecally and more to the right. Inspection extrathecally was incomplete as intense venous congestion caused bleeding so that a venous malformation was suspected by the surgeon, who considered it was impossible to proceed. The posterior decompression and section of the dentate ligaments, it was hoped, would relieve the symptoms.

Catheterization was required for five days, after which the bladder was normal. Other symptoms improved. When discharged on November 26, 1949, there was mild pain only in the right arm, and the patient felt movements of the right fingers and foot to be freer. There was subjective improvement until April, 1950, when symptoms recurred and steadily became worse. She was re-admitted in February, 1951, and refused conservative treatment. The left arm was now involved to a greater degree and there was pain on the right in the middle finger, on the left in the fourth and fifth fingers. The right leg was weaker and apt to fall suddenly. Pain was burning in nature. Both hands and the right foot sweated freely.

On examination it was seen that the same muscles of the right upper limb were affected, but they were weaker and more wasted. There was now some wasting of the intrinsic muscles of the left hand with weakness of triceps and biceps. The right leg's condition was as before. The deep reflexes on the right upper limb were now slightly increased, and depressed on the left. There was clonus at the knee and ankle on right, with slight hyper-reflexia of the left leg. The right plantar response was extensor, the left flexor, and the abdominal responses were absent. There was diminished sensation to pin prick and cotton wool in C.6, 7, 8, and T.1 right, maximum C.7. Vibration and joint sense were markedly depressed in the right arm but intact in the left. Vibration was slightly diminished in the right ankle. Thus there was a marked increase of abnormal neurological signs.

Radiographs showed loss of cervical lordosis, exaggerating the prominence of the spurs compared with 1949.

Second Operation.—On February 22, 1951, the old incision was re-opened and lamina C.6 removed. The dura was re-opened. The cord was very pale and stretched over an area corresponding with the body of C.5. A large anterior spur was found at C.5–6 level with a "high-spot" indenting the cord medially and minimal in height laterally beneath the roots. A lesser spur was seen at C.4–5. The spur at C.5–6 was removed completely extra-thecally after great difficulty with extreme venous haemorrhage. No venous malformation was seen. The cerebrospinal fluid now flowed freely, the cord was in a more normal position with improved capillary pattern posteriorly and lessening of venous congestion. The dura was closed.

The patient was discharged on March 31, 1951. She experienced a slight increase of numbness at C.7, but no other neurological change. She had passed urine normally on the first post-operative day.

On August 7, 1951, she had no pain or paraesthesiae in either arm, but some numbness of the ulnar side of the right hand. Movements of the arms and legs were free. She could write legibly and sew. Her walking was much better and the wasted muscles were filling out. There was a slight increase in the left biceps jerk; otherwise upper limb reflexes were normal. The right plantar was now flexor.

She experienced no loss of vibration or joint sense, but slight diminution of reaction to pin prick in C.6 and 7 right, especially in the tips of the fingers.

This case is possibly the most important of the series for it shows not only that an anteriorly placed, transverse bony spur, unassociated with any protrusion of disc material, can produce serious malfunction of both spinal roots and cord, but it also points the surgical moral that when the distorting abnormality is placed in front of the cord it is futile to expect permanent relief from posterior decompression with section of the dentate ligaments.

In fact it is more probable that distortion of the cord with aggravation of symptoms will result thereby.

Discussion

Bony " spurs " were removed surgically from a series of 19 cases. All but one of these spurs were in the cervical region. Eight illustrative cases are quoted to show the variability of the neurological syndrome encountered and, within the limits of a perhaps inadequate follow-up period, to record the amelioration to be expected from a surgical attack on these abnormalities. I would again emphasize that all these cases had previously had conservative treatment. This is a relatively new field in surgical neurology and this paper must, therefore, necessarily be in the nature of a preliminary communication. As further experience is gained, so one must expect modifications in approach. It is with a full realization of these considerations that I now propose to discuss the various features of this series of cases.

Aetiology.—The youngest patient was 27 and the oldest 73. Six were in the third decade, ten in the fourth, and one in the sixth.

Generalized osteo-arthritis was present in only the three oldest patients, but in the rest the spur or spurs were isolated radiological lesions. It is for this reason that the term bony " spur " is used in place of osteophyte, for the latter term conjures up a picture of widespread osteo-arthritic change.

In 17 cases an episode of sudden movement of the neck was remembered by the patient, but was often not related by them to the onset of the symptoms, which appeared in some patients months or years later. In others the onset of symptoms coincided with the trauma and the mature spur was shown to be present immediately after.

Form and Nature of the Spur.—The *x*-ray appearances of the spurs have been described by Turner and Oppenheimer (1936), and Philip (1950) has given diagrams showing the lipping from the margin of a vertebral body. The silhouette of the cross-section of the spur is *more often* that of a double lip from the adjacent body margins of two vertebrae and each contributes an equal share. The bases arise from varying areas and the cusps taper and curve to meet each other at the apices, $\frac{1}{8}$ in. to $\frac{3}{16}$ in. beyond the normal body contours. If the bases are large, they appear as two pyramids, each with perpendicular or curved edge (Fig. 1). The pyramids may be of denser contrast than the normal bone. In many the base is small and the cusp long and a " gargoyle " appearance may result. In others a wider base with a rounded apex show them as vague rounded hummocks. Usually, however, the cusps are separated in the same line as the interbody space.

FIG. 1.—Line drawing to show diagrammatically the profile projections of the commoner shapes of the bicuspid spurs as seen radiologically.

At operation the linear extent of the spur formation is realized. It extends across the whole length of the posterior margin of the body (or bodies) from within one intervertebral foramen to the other. It is usually bicuspid at any point of cross section. Its side-to-side elevation is like a suspension bridge with two " high-spots " just within the lateral margins of the cord (Fig. 2*b*). Sometimes there is but one " high-spot " in the midline and here there is little ridging laterally towards the intervertebral canals and little compression of the emerging mixed nerve roots. The extent, height, and position of the spurs is usually only fully realized at operation and it is impossible to gauge their size by radiographs alone.

The spur has a brittle, sugary consistence. The bone is cancellous within a thin shell and scrapes away from its base on the body, leaving a shiny cleavage line. These features would suggest that it is no mere eburnation of the body margin, but is an ossifying structure, capable of considerable growth. The posterior common ligament, stripped from the body, covers the apex of the spur. There is a definite cleavage line between it and the bony spur. The ligament may hypertrophy over the apex to a thickness of $\frac{1}{16}$ in. or more, and thus add its thickness to the height of the spur. The annulus fibrosus retains its normal contour between the body margins.

As the apices of the cusps are strongly opposed and as the firm annulus fibres bind their bases, it is inconceivable that disc material protrudes between them. The disc material would appear to have atrophied *in situ*.

Seldom is the spur formation at one level or on one side alone. Usually the formations are at more than one level although they may vary in size. More often bilateral, the spurs may be asymmetrical, and then may show up on one side only radiologically.

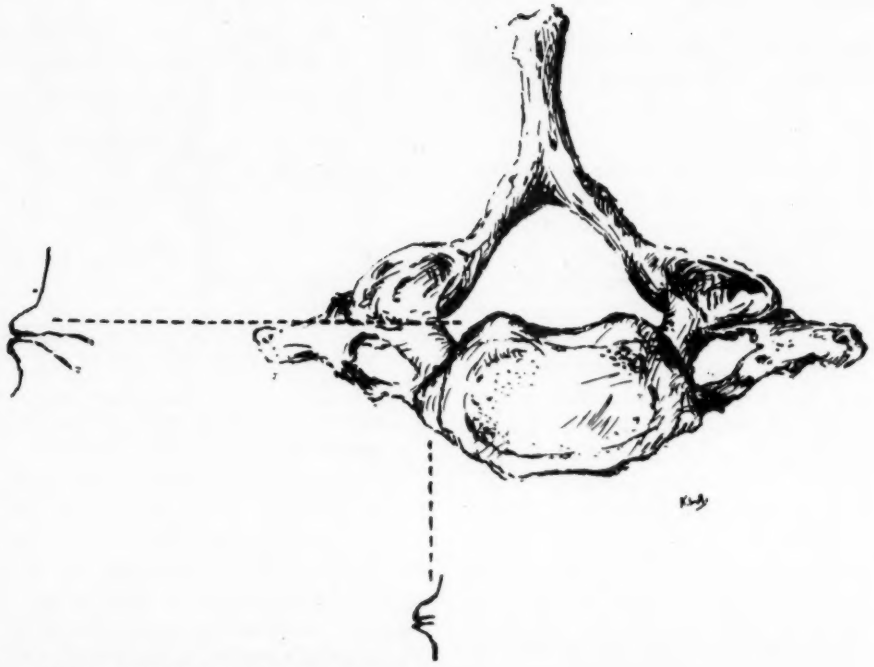


FIG. 2.—Drawings of cervical vertebrae :—
(*a*) Cervical 7 to show spur position and profile projection in the lateral and antero-posterior views. corresponding to *x*-ray plates Nos. 3a, 3b, and 4.

FIG. 3*a*.—Right oblique view to show " pyramid " type of spur projecting into the intervertebral canal of C.5–6.

FIG. 3*b*.—Antero-posterior view corresponding with Fig. 3*a* to show the lateral extension of the spur into the vertebrarterial canal.

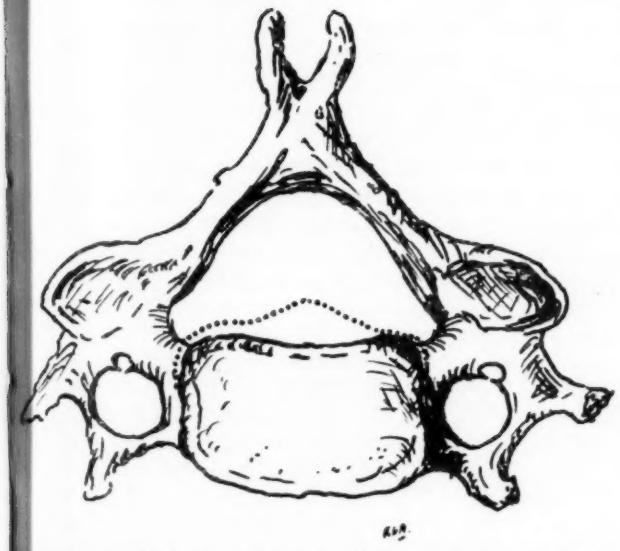


FIG. 2c.—Cervical 5 to indicate the osseous bridge extending across the vertebral canal as in (*b*). Here the high spot is single and mesial in position having an anterior relationship more to the cord than to the roots.

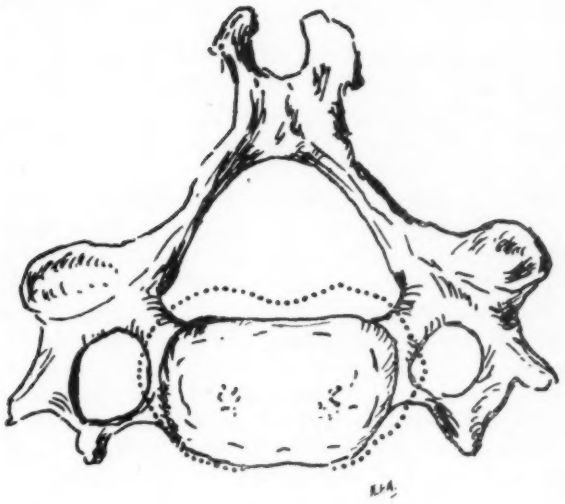


FIG. 2*b*.—Cervical 6 to show diagrammatically by the dotted line the annular zone of osteophyte formation. The spurs are indicated with two high spots, each just within the lateral margin of the spinal canal. The more lateral extension is through the intervertebral nerve canal and is even encroaching into the vertebrarterial canal.

FIG. 4.—Lateral view to show typical bicuspid spur with interspace narrowing at C.5 on C.6. The cervical lordosis is straightened at this level.

Relationship of Spurs to Adjacent Nervous Structures and Effect on Nervous Function.—A bridging spur at a single level in its entire linear extent lies anterior to all nervous structures. It follows that from right to left it is related to the right sympathetic nerves in the vertebral arterial canal, the plexus from the lower cervical ganglion (Fraser, 1940), the emerging right mixed nerve in its dural sleeve, the cord and the right and left anterior root fibres destined to emerge at the level below (Fig. 5), the left mixed nerve and the left sympathetic nerves.

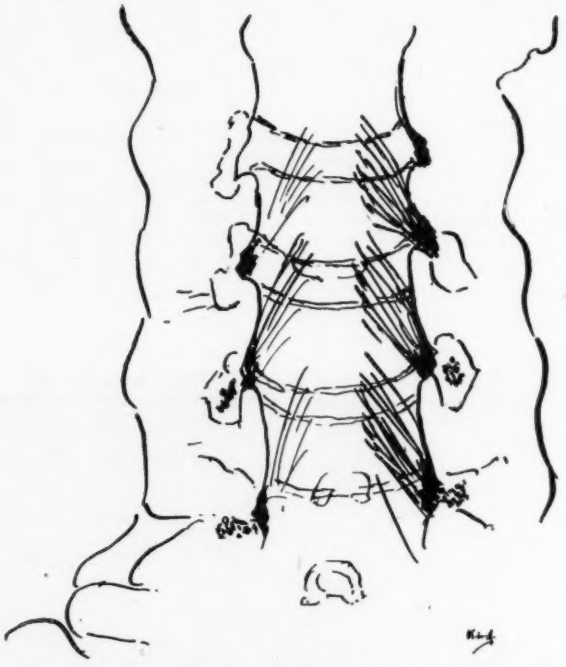


FIG. 5.—Line copy of *x*-ray plate to bring out the relationship of the radicals of the emergent root to the interspace at a level one higher.

If there are spur formations at two adjacent levels, at the lower level the pattern is repeated, but the anterior root fibres already involved at the higher level are again affected within the mixed roots, emerging at the lower.

At operation, when the dura is opened, the cord is found kinked and displaced backwards over a short distance, which corresponds to the anteriorly placed spur. The posterior surface of the cord is pressed against the dura, which is often thinned over it. The cord is pale and immobile and may even be flattened from before backwards. Transmission of pulsation by the cerebrospinal fluid over the affected segments is blocked but appears again below. The appearance of the affected area is as if it were "frozen". The displacement of the cord and the accompanying vascular effects are exaggerated by passive flexion of the head and neck. Indeed at operation the flattening down of the nervous structures upon the anterior bridge may be so great as to prevent any access or retraction unless adequate posturing ensures a maximum cervical lordosis. In the more severe deformities as flexion is increased the "freezing" spreads centrifugally and the roots at nearby levels take up the tension. Above and below the spur there is congestion of the anterior extrathecal plexus of veins. At the apex of the bridge they are obliterated. A free flow of venous blood in these channels is re-established after the spur is removed. I do not wish to imply that disruption of venous or arterial flow is complete or constant in any but the most severe deformities. In all cases, however, there may be partial and temporary disruption proportional to the range of neck movement.

Clinical Picture.—The cases can be grouped into (1) those with cervical root involvement only. These patients almost invariably have pain in one or both upper limbs and usually there is nerve conduction loss. This almost always involves more than one root. (2) This group has involvement of the cervical cord, with conduction loss in the trunk and lower extremities. In the more severe cases and in the later stages both root and cord signs and symptoms may be present. As a rule the cases fall into one of the above two groups. Operation may be indicated in either group.

In this series the younger group—mainly the third and fourth decade—belongs initially or entirely to group 1. The older group belongs to group 2.

Group 1.—Pain is the initial and usually the dominating symptom. Frequently on one side only, it may spread to the other. The patients have difficulty in defining it, but it is usually severe. It is most frequently situated in the shoulder, scapula or upper thorax, pre-axilla or precordium. At this stage attempts to identify its origin within a certain root are impossible (compare Philip's findings). Later it will radiate into the arm; rarely does it enter any consistent finger distribution. The paraesthesiae which follow are of greater aid in root localization. These are often brought on by neck movements and felt in an index finger, or a definite diagnostic combination of fingers. Later sensory dermatome loss may be elicited. This is frequently circumscribed at first, but unfortunately often variable. More insidiously muscle wasting appears, corresponding to the affected roots. Fluctuating autonomic changes are often seen and may be confined to one aspect of one forearm or hand. To sum up: pain is of little localizing value, paraesthesiae are of slightly more value, dermatome

loss is of great help, and finally motor loss is critical to the roots involved.

In some cases there is an extraordinary dissociation between sensory and motor signs in their dominance of the clinical picture, despite the invariable involvement of the mixed nerve root in these cases. This is clearly shown by contrasting Case 3 with Case 5. The most probable explanation is afforded by postulating a vascular deficiency as opposed to a pure mechanical compression of the nervous elements. At operation, moreover, it has been consistently found that the mixed nerve root, immobilized over the bony spur, is extremely " touchy " and twitchings of the muscles innervated by it occur with very slight mechanical or electrical stimulation. The most irritable portion in terms of motor response is the antero-inferior aspect of the nerve, which is nipped and attenuated to a greater extent in those cases where muscle loss predominates. This differential anatomical nipping depends in turn upon the pre- or post-fixation of the emerging mixed nerve, relative to the spur. These observations may provide some explanation of the unpredictable neurological effects.

The state of the reflex arcs is often extremely variable in these cases. In some patients the arm reflexes are increased, despite obvious muscle wasting. In others they are reduced early and later lost. In Case 1 in 1945 reflex diminution was reported from the National Hospital, London, but in 1949 the same reflexes were increased, although muscle wasting and weakness had increased. Fasciculation was present in all cases with muscle wasting. Frequently there is a very sudden exacerbation of symptoms, particularly in the motor sphere.

Group 2.—Pain is rarely evident. The most frequent disability is referable to the pyramidal tracts. At first the symptoms remit. Later there is progressive weakness and stiffness of the legs, with bilateral Babinski responses. Over a period of months the motor weakness gradually creeps upwards. Some patients, more particularly the aged, show no sensory loss. This overall picture would suggest a systemic degenerative pathology, particularly where a lower motor neuron lesion in the arm appears. In such a case a diagnosis of progressive muscular atrophy would be reasonable. In one case the lower motor neuron lesion occurred before the involvement of the pyramidal tracts. In another case, No. 6, the spino-thalamic tracts were early involved, whereas the motor symptoms were more subjective than objective. Here the spino-thalamic tract of one side only was involved.

Clearly impairment of any tract or combination of tracts can occur. Often a remarkable and very crisp tract dissociation occurs initially and may persist throughout. This selectivity is explicable on a vascular rather than a compressive basis. Compression is often not great, nor does it remain focal.

Case 4 illustrates a diagnostic hazard of importance. At first an ill-defined pyramidal lesion indicated no certain upper level. Only later root signs in the upper limb appeared and even then there was a " gap " between root and tract levels. This " gap " narrowed and eventually the levels coincided at the site of the spur. Premature assessment led to a useless laminectomy. In instances therefore where no definite lipiodol hold-up can be shown, and where the upper level, sensory or motor, fluctuates, surgery should be withheld until the level becomes definitive and stable.

Autonomic changes, such as increased sweating of the hands, frequently accompany the other disabilities. There may be coldness of the legs, often not noticed until after operation, when the patient will say, " Doctor, my legs are warmer. I did not know how much they were affected ". This symptom may be the only one suggestive of cord involvement in the cases falling apparently into Group 1.

Diagnosis.—This rests on both clinical and radiographic evidence. At first positive radiographic findings were considered essential for the diagnosis. This still holds true where there is mixed root involvement. In some cases, where cord signs predominate, the spur has not been demonstrated convincingly radiologically. This is because it is not always easy to obtain the exact centring of the rays to throw up the spur profile, especially when the " high-spot " is medial. Frequently this " fugitive " spur does not coincide with the obvious narrowed interspace.

Even where no radiological evidence of spur is shown and even when a narrowed interspace is present, the existence of adequate clinical evidence of root involvement is more likely to indicate spur formation than disc prolapse.

Myelography was unnecessary in all but three cases. In these there was suspicion of tumour or angiomatous malformation. In these cases myelography was for exclusion only. It gave no other essential information but confirmed the presence of the anterior bony spur. In the A.P. projection a " flat " cupola corresponding to the interspace was produced with both upward and downward flow of the opaque medium with the patient in the prone position. Tilting must be extremely slow or the " spill-over " will be too rapid for the recognition of the deformity, and this will be missed if the

patient is placed even with minimal obliquity. Lifting of the opaque medium over the spur will show on the lateral view and this is perhaps more useful than the A.P. viewing. Lipiodol (Lafay), 1–2 ml., shows the abnormalities more adequately than less viscid media.

Differential Diagnosis.—Case 1 shows the similarity to motor neuron disease. Pain was a little severe and the progression too slow. The history of trauma is significant. Fasciculation was seen in the atrophied muscles.

Case 2 with its age of onset, hypochlorhydria, and the suspicion of remission suggested subacute combined degeneration of the cord.

A confident diagnosis of angiomatous malformation of the cord was made in Case 6. This pathology was in fact present, but the onset of root signs and the alleviation of symptoms after operation points to the bony spur as being mainly responsible for the syndrome.

X-ray and clinical evidence serves always to distinguish from conditions affecting the more peripheral parts of the brachial plexus.

Conservative Treatment and Indications for Surgery.—On routine radiographs of the spine spurs are frequently seen. Where there are no symptoms these mean little, except that the reserve is sufficient to allow of accommodation of the nervous tissues. Naturally no treatment is required. They are a potential danger only.

In a second group of patients there are symptoms of general tiredness and aching throughout the spine. There is minimal clinical evidence of neurological involvement. Radiographs of the whole spine are taken and an incidental finding is a cervical spur, usually between C.5–6 or C.6–7. It is this type of case about which so little is known, but one supposes that just as a lumbo-sacral disc degeneration may in many cases cause chronic back disability, so a cervical spur may represent the weak spot in the mechanics of the spine, particularly where there are abnormal postural strains. These strains would theoretically be most liable to occur where high mobility changes to low, that is at the cervico-dorsal junction and between C.5–6 and C.6–7. Treatment of these cases aims at correction of the postural abnormalities and to this the majority respond.

In cases of this type, where posture plays such an important part, drooping of the shoulder girdle may well supervene, and weakness of the scalene group of muscles will result in pull and drag on the brachial plexus, this in turn being transmitted to the spinal roots, which then impinge upon the spur as "a rope upon a jagged pulley". Such an effect is particularly liable to be produced in states of chronic ill-health where muscle tone is below par or where prolonged immobilization in collar, plaster, or traction allows the muscles to lose their inherent tone. When this muscle state exists a relatively minor strain or injury will produce the neurological syndrome, dependent on compression by the bony spur.

Conservative treatment should aim to avoid this reduction of muscle tone and should not therefore include the prolonged use of collars, plasters, or unduly prolonged neck traction. Where possible it should allow of mild muscle activity and avoid extreme disuse of the anatomical splinting muscles of the neck. Prolonged, complete immobilization, during which neck muscles atrophy or become lax, is contraindicated. I would, however, emphasize that conservative treatment, where these principles are borne in mind, results in complete alleviation of symptoms in the majority of cases, at least temporarily.

The third group of patients is that in which all conservative treatment has failed to stem the progress of the symptoms and signs. In this group it is to be noted how rapidly the neurological syndrome may progress and particular attention is to be paid to the development of cord signs. All the cases presented in this paper fall into this group. None was operated upon until it was evident that adequate conservative treatment had failed. It is further emphasized that prolonged disability may be prevented by timely surgical intervention. The size of the spur, as shown radiologically, is no indication for surgery when divorced from the clinical state.

Surgical Technique.—The aim is to obtain the fullest decompression of nervous tissue, and to restore the normal mobility of this tissue in relation to the dura and to the extradural surroundings. The effect of the spur is not entirely one of compression. It fixes the dura over a wide area, much as a pelt pegged over an uneven surface. The peripheral points of fixation are the dural sleeves of the emergent roots in the intervertebral canals of each side, usually at more than one level. Relief of symptoms is proportional to the re-establishment of the normal soft-tissue "glide". Thereby the cerebrospinal flow and the vascularity of nerve roots and cord are improved and the extrathecal venous plexus disengorged. This last engorgement may be so extreme before removal of the spur as to suggest venous malformation and to have prevented adequate surgery before (Case 8). The anterior spur is removed to an extent that will ensure this "glide" and reconstitute the fluid spaces throughout the range of normal neck movements.

The patient is postured prone on a table fitted with shoulder and adjustable horse-shoe head rests. Freedom of respiratory excursion is ensured. The head is symmetrically placed forehead down with a fairly full degree of flexion of chin to chest. Local subcutaneous infiltration of novocaine is used and the incision made in the midline from the external occipital protuberance to the vertebra prominens, the lower extremity being adjustable to the site of the spur. The large spine of C.2 is brought into the field to serve as a definite landmark but need not be fully dissected from the muscle. The laminae on both sides of the midline are exposed by means of the usual endothermy cutting technique with coagulation haemostasis.

Care must be taken to avoid tearing or cutting the posterior primary motor division. Light, cerebellar, self-retaining retractors are preferred to bulkier types, which retract mainly at depth. Manoeuvres later in the operation require that the field be not obscured by the bulkier, grid type of retractor blades.

The extent of the lamina removal varies with each case. In general, where root involvement is predominant, a " window " technique can be used. To achieve this the simplest method is the use of a $\frac{5}{8}$ in. burr centred equally on two adjacent laminae, the opening being placed laterally. Exposed ligamentum flavum is dissected away with a knife to reveal dura. Such openings can be placed at the same level on each side to facilitate removal of the spur from each side (Fig. 6). In cases where spurs are at two adjacent levels it is more rational to remove a whole lamina than to make four burr holes. This naturally applies to all cases where there is cord involvement and the dura is required to be opened. The upper and lower borders of adjacent laminae are trimmed with rongeurs to expose the upper and lower " axillae " of the respective nerve roots.

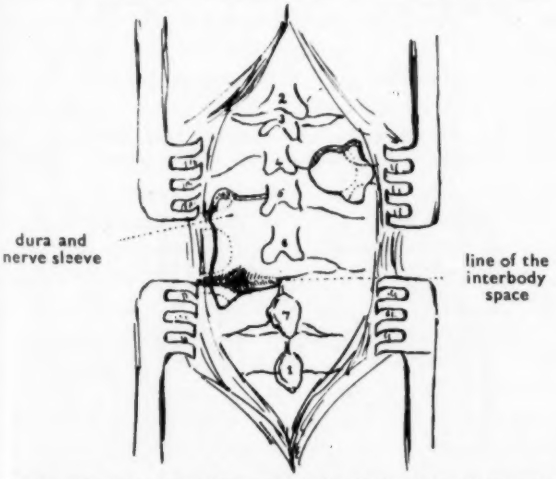


FIG. 6.—Sketch to show diagrammatically the " window " method of laminectomy approach to a single nerve root, or the more extensive method of exposing two adjacent levels. Either exposure may have to be used on both sides. The upper and lower axillae of the roots are exposed. The cross-hatching indicates the position of the spur on the one side relative to the overlying mixed nerve root and the cord.

The head is moved through as full a range as possible to assess the degree of tightening and fixation of the dura and neural tissues and is then left in the position of maximum laxity.

A fine blunt dissector is introduced into the nerve foramina and the upper and lower nerve axillae to feel for the anterior deformity. This inspection is carried out on both sides so that the " high-spots " may be recognized. Before it is possible with safety to retract the flat, taut dural nerve sleeve, some of the posterior lip of the nerve canal must be trimmed away. The anterior deformity is then approached through the lower axilla, the spur being felt to rise rapidly to its full height quite medially, just within the lateral border of the cord. At this stage the dura must *not* be forcibly retracted over the high spot. The engorged extrathecal veins now begin to bleed and small, stringed " patties " are tucked gently between dura and bone to control them. The posterior common ligament is exposed and, stretched over the spur, it resembles very closely the tense, pale bulge of a prolapsed disc. Even on palpation it may be elastic and springy and this " give " is due to the fact that it is usually hypertrophied. As the ligament is incised crucially, the bony spur is easily seen and felt. From the point of incision the ligament is scraped away from the ridge of the spur for about $\frac{1}{16}$ in. in all directions.

Using a thin $\frac{1}{8}$ in. chisel with a slight but firm downward pressure combined with a rotary motion, a shaft is rapidly made through the exposed portions of the spur (Fig. 7). This ends upon a glistening base-plate, formed by the normal body contour. This must not be perforated. The shaft is enlarged centrifugally and the dural tension is thus freed to allow very much lighter retraction and, as the excavation of the spur by scraping and paring proceeds laterally into the nerve canal and medially beneath the cord, retraction may be dispensed with altogether. *At no time is there excessive medial retraction on the cord.* Instead the little chisel, with its very slightly curved end, is used with a progressively more acute angle to the horizontal plane. Provided the muscle and skin at the sides of the wound can give, the chisel can be used at such an angle that its tip will cross the midline anterior to the cord. With each " scrape " the chisel tip eases aside the posterior common ligament to avoid buttonholing it or nipping the dura. In this way the lower lip of the spur is removed on one side of the

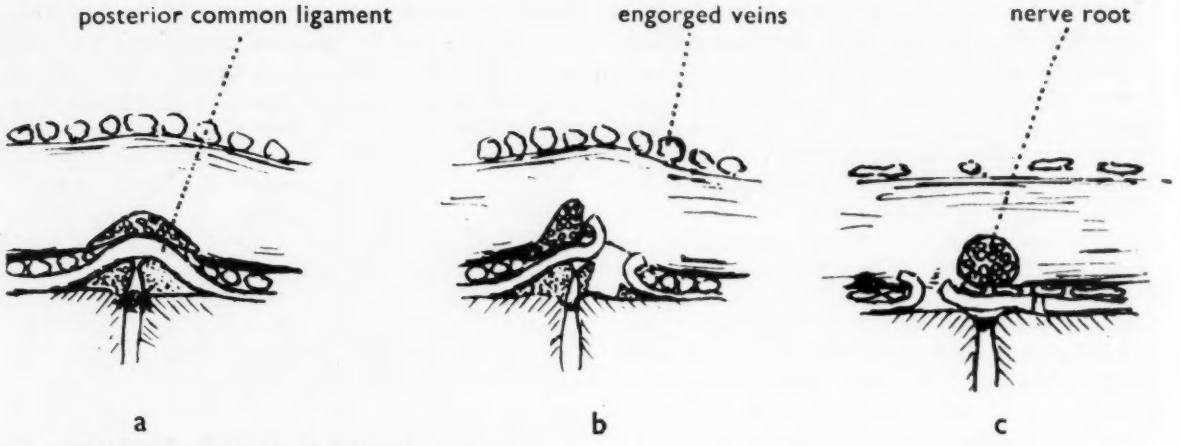


FIG. 7.—Serial diagrams indicating in longitudinal section :—

(*a*) The dislocation of the cord and root over the bicuspid spur and the thickened posterior common ligament interposed between them.

(*b*) The retraction of the root at its lower edge, incision of the posterior common ligament, and the shaft sunk through the lower lip of the spur.

(*c*) Upper and lower lip of spur removed, all soft tissues reconstituted in normal positions.

The posterior body contour and annulus fibrosus is unbroken and engorgement of extrathecal veins relieved.

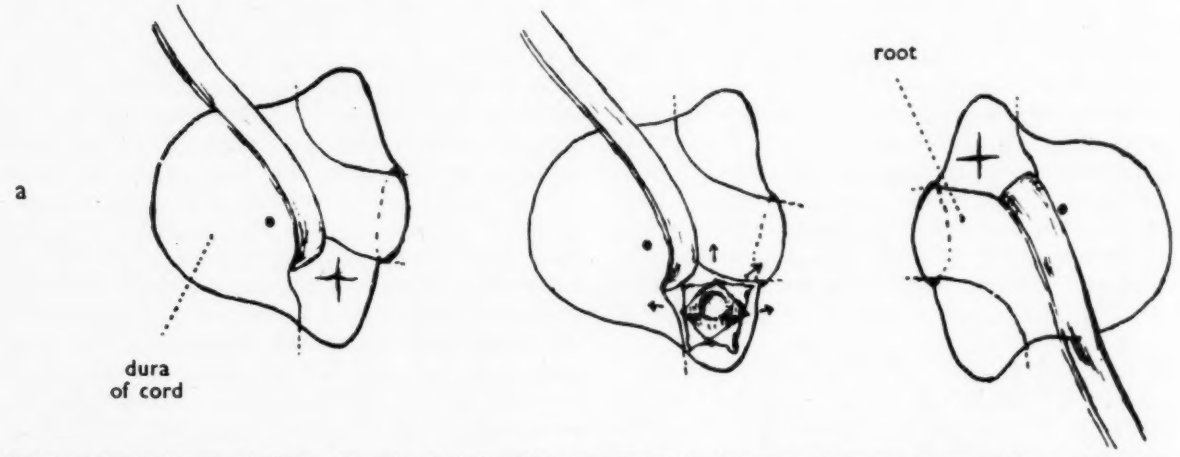
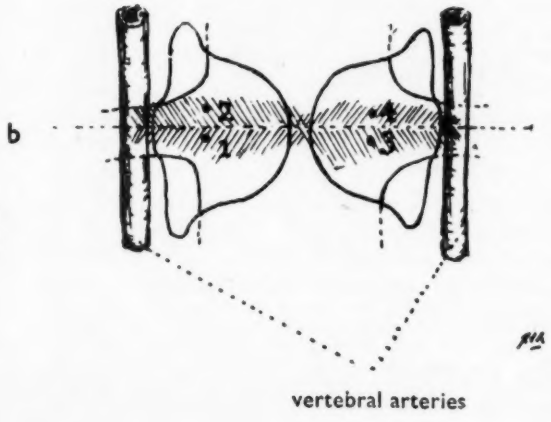


FIG. 8.—(*a*) Serial diagrams indicating through a " window " the retraction within the lower and upper nerve root axillae and the cruciate incision through the posterior common ligament and the shaft into the spur (corresponding to Fig. 7(*b*)), from which excavation is carried centrifugally.



(*b*) Cross-hatching indicates the total spur bridge divided diagrammatically into four quadrants each to be removed in turn from the corresponding axillary approach.

midline. The process is repeated from the upper axilla to remove the upper lip. The interbody space is not entered but can be felt as a depression after the spur has gone. The procedure is carried out from the other side at the same level. The dura is opened and after cutting one or more dentate ligaments on one or both sides transthecal inspection is made. Any remaining spicule or roughness is removed, still by the extrathecal route. The dura is not incised anteriorly and therefore transthecal removal cannot be used. In the spurs with two lateral " high-spots " removal to the midline is not necessary, but in the median spur it is essential.

The floor of the spur's cavity is left as the glistening body contour above and below the interspace. The posterior common ligament is virtually intact, but bridging across the cavity. Occasionally hypertrophy of the ligament is so extreme as to continue posterior displacement of the cord. It then has to be trimmed, but if possible it should be kept in continuity.

In the first cases the dura was opened early, before the spur had been dealt with, in order to assess the degree of cord displacement and compression. The cord was found kinked and pushed against the posterior dura, which was often so thinned that the cord surface could be seen through it. The kink coincided with the spur and the posterior surface of the cord showed marked pallor for some distance above and below. In most cases flexion of the neck accentuated the kink and pallor but in one case the effect was from extension.

The sufficiency of the anterior, extrathecal decompression is assessed by the elimination of the kink and the re-establishment of a normal vascular pattern of the cord throughout a full range of neck movement. The engorgement of the extrathecal venous plexus quite suddenly disappears and it is reasonable to assume that this coincides with adequate release from the abnormal tensions. Usually engorgement persists until all levels involved in spur formation are dealt with. It has, however, happened that removal of the major spur has so eased tensions over several segments that the minor spurs were not touched (Case 6). The wisdom of this is doubtful in view of the residual symptoms in this case, referable to the adjacent segment.

Finally a thin layer of " gel-foam " is lightly tucked between the bone and the posterior common ligament to promote haemostasis. It is also placed about the nerve sleeves, where it has not been possible to preserve the normal venous or fatty cushion around the sleeve. The cerebrospinal fluid is replaced with warm Ringer-Locke solution and the dura closed with interrupted black silk sutures on an eyeless, curved, non-cutting needle. The pia-arachnoid over the cord is not disrupted at any stage.

Haemostasis is meticulous, for scar tissue arising within excess blood clot around the nerve sleeves and dura would render pointless an operation, which is not easy neurosurgically, and replace one form of fixation or compression by another. Muscle and fascial layers are closed in individual layers, no dead space is left, and the wound is made watertight. White, Japanese silk is used for tension sutures and one layer of double black silk in the first muscle layer above the dura acts as a marker, should a later re-opening be required. The skin is closed in two layers. A vaseline dressing is applied, backed with cotton wool and gauze and the whole sealed with elastoplast strips, applied to prevent as far as possible splaying of the scapulae. No plaster of paris splintage is necessary.

The patient is nursed lying on his back on a fracture board for the first 10 days. Catheterization has never been necessary after 48 hours. Leg movements are permitted from the start and aided by mild massage and guided exercises.

Superficial skin sutures are removed on the tenth to twelfth day and the patient then allowed to roll himself from side to side. He sits up in bed on the thirteenth day, hangs his legs over the edge of the bed on the fifteenth day, and thereafter is allowed up for gradually increasing periods. Immediate relief of root pain is the rule post-operatively, but in many instances it returned after a few days, and even was accentuated for about one week. The pain gradually lessens and may re-appear on and off, only as a fleeting, disagreeable sensation, for some months before disappearing completely.

Conclusions and Summary

Characteristic cases from a series of 19, operated upon since April, 1949, are reported. From these cervical osseous spurs, anterior to the nerves and cord from side to side, have been removed. Such spurs have been recognized for some time, but their major importance in the production of neurological syndromes has not been sufficiently stressed.

In my experience prolapsed cervical disc is extremely rare and when present contributes less to the disability than the osseous spur which invariably accompanies it. In all cases reported here the symptoms were attributable to the spur.

Symptoms may be due to root or cord involvement by the spur, separately or in combination. With root involvement pain, with or without neurological signs, is always present. With cord involvement insidious, bizarre pictures with long-tract deficit arise in the trunk and lower limbs In both types, although complex structures are in-

volved, crisply dissociated pathological effects may persist throughout the course of a progressive illness.

The striking similarity of some cord syndromes to progressive muscular atrophy and subacute combined degeneration of the cord is pointed out.

The term "spur" is preferred to "osteophyte". The spur bridges across anterior to the nervous tissues and should be removed entirely throughout its length. Reorganization of the normal anterior "glide" is the aim of the operation. Posterior laminectomy is considered insufficient.

Surgical treatment is undertaken only after conservative treatment has failed, and where the neurological disability is becoming steadily or rapidly progressive.

Removal of the anterior spur has proved safe with the technique described.

In work which evolves slowly, step by step, to a stage of fulfilment, the individual identity of contributing personalities tends to fade, but not so the value of the help and criticism of each member of our neurosurgical team. In this spirit I should like to thank my colleagues. I thank Mr. R. A. Krynauw especially for his guidance and example, and particularly for his report on Case No. 7, and Dr. Andrew Watt for his help with the manuscript and his criticism. I received encouragement from Sir Hugh Cairns, to whom I had the opportunity to present a series of post-operative cases and to demonstrate the technique of the operation during his visit to South Africa in August, 1951.

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J. Neurol. Neurosurg. Psychiat., 1952, **15**, 37.

TRIDIONE COMPARED WITH MALIDONE IN THE TREATMENT OF PETIT MAL*

BY

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Tridione (3,5,5,—trimethyloxazolidine—2 : 4—dione) has been extensively used in the treatment of petit mal for five years, and its success in more than 60% of cases has been well established. Its dangers and limitations are also well recognized (Richards and Everett, 1944 ; Lennox, 1947).

During the past two years 25 men between the ages of 17 and 52 who suffered from frequent petit mal, and 15 of whom had previously been treated with tridione, were given a similar substance, namely " malidone " (3-allyl-5-methyloxazolidine-2 : 4-dione), in an attempt to compare the relative merits of the two drugs.

The average dosage was one capsule (0·3 g.) thrice daily. The results are compared with those obtained in a previous series of 35 cases treated with tridione (Table I).

TABLE I

COMPARISON OF RESULTS OF TREATMENT OF PETIT MAL BY TRIDIONE AND MALIDONE

	Tridione (35 cases)	Malidone (25 cases)
Petit mal completely or almost completely controlled	25%	20%
Petit mal attacks reduced by half or more than half	37%	44%
Slightly benefited	12%	16%
No change	20%	16%
Worse	6%	4%

As will be seen from this table, the results are closely comparable as far as the control of petit mal is concerned.

Previous experience in the tridione series in this hospital had shown that toxic reactions are less likely to be produced if the drug is introduced gradually, and for this reason I have advised an initial dose of one capsule daily for several days, this being increased to the optimum number of capsules which without toxic reactions will effect the greatest reduction in the number of attacks. Similar caution in using malidone would appear to be advisable.

Table II compares the incidence of toxic reactions produced by the two drugs.

TABLE II

INCIDENCE OF TOXIC SYMPTOMS AND SIGNS

Symptom	Tridione	Malidone
Photophobia	80%	Nil
Drowsiness	22%	8%
Restlessness and lack of concentration	4%	4%
Slight ataxia	8%	Nil
Incoordination of movements of hands	Nil	4%
Hiccup	8%	Nil
Slight epigastric discomfort	8%	Nil
Skin rash	8%	12%
Marked leucopenia	4%	4%
Increase in attacks of grand mal	3%	4%

The most striking differences are the absence of photophobia and the relative infrequency of drowsiness in the malidone series. Photophobia, or " the glare phenomenon ", in patients taking tridione, may be sufficiently severe to necessitate the wearing of dark glasses. Although tolerance has been found to be acquired in the majority of cases after a

* Communication to the International League against Epilepsy in January, 1951.

few weeks, it is obviously an advantage to have a drug which does not produce a symptom sufficiently trying to deter some patients from persevering with the treatment. Malidone also appears in this series to have the advantage over tridione in not producing ataxia or gastric irritation.

Summary

Twenty-five patients with petit mal, 15 of whom had previously been taking tridione, were treated during the past two years with " malidone ".

The therapeutic results with malidone were broadly similar to those obtained with tridione, 80% of the patients deriving benefit.

Photophobia is common in patients taking tridione (80%) but malidone did not produce photophobia in any case in this series. Other toxic symptoms or signs were usually less severe and more transitory than those experienced in the series treated with tridione.

I wish to thank Messrs. British Schering, Limited, for generous supplies of " malidone ", and the British Schering Research Institute for their biological report on various derivatives of oxazolidine—2 : 4—dione.

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J. Neurol. Neurosurg. Psychiat., 1952, **15**, 39.

COGNITIVE FACTORS IN EPILEPSY

BY

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A considerable number of studies has been carried out on cognitive aspects of epilepsy. Most of these have dealt with the general mental ability of the epileptic patient; for example, Sullivan and Gahagan's (1935) study in which of 103 epileptic children tested 14 were found to be mentally defective, the median I.Q. was 92, and I.Q.s ranged from 11 to 141. Sheps (1947) has commented on the widely varied type of patient selected, and says, "This bias in the material is reflected in the wide variations of the results; for example there is a range in the reported mean I.Q.s of from 113·5 to 78." Sheps, studying the intelligence test results of the entire group of male epileptics of military age who were not in institutions for one military district in Canada from 1942 onwards, found their intelligence only slightly but significantly lower than that of the general population; the increase in the number of mental defectives in the epileptic group, 8·94% as against 7·29% in the total group of recruits, was not statistically significant. He points out that "most of the defective epileptics are institutionalized by the time they reach military age".

Other investigators have dealt with the general mental ability of epileptics more from the point of view of deterioration. Lennox (1944), quoting from his own findings in 1941 on a group of 1,640 clinic and private patients, states that

> ". . . 24% were found to be abnormal during the first years of the disorder and 54% abnormal if the disease had persisted more than 25 years. There is progressive decrease in mentality with the total number of attacks, especially if these are psychomotor or grand mal or a combination of these. Cases with pure petit mal may go unscathed."

In addition a number of studies has been made to test directly the evidence for deterioration by giving groups of patients tests over a period of years. Lennox in commenting on these studies says, "Unfortunately the results are distorted by the fact that not all the original patients are followed throughout the period."

The further question of whether or not there are any special mental abilities in which the epileptic is inferior has not been overlooked. Somerfeld-Ziskind and Ziskind (1940) studying a group of 100 epileptic out-patients found memory retention and language ability somewhat defective. Arluck (1941), whose group numbered only 16 out-patients, reported results of the picture absurdity test being significantly worse in epileptics than in control groups. Lennox and Collins (1945) reported that epileptics who were given the Wechsler-Bellevue tests did relatively poorly in the performance tests. Capps (1939), who gave a number of word-meaning and usage tests to idiopathic epileptics, whose mental deterioration had been clinically estimated, found that "all of the vocabulary tests show a consistent, progressive and reliable tendency towards a reduction in the mean scores associated with the group of subjects showing the greater degree of mental deterioration." Patients on admission had an average I.Q. of 91·1 but on the Terman vocabulary test their I.Q. was only 53, and he concluded "Terman Vocabulary does not remain constant and unchanged with deterioration in idiopathic epilepsy and therefore cannot be employed to determine the native intellectual capacity of deteriorated epileptics."

All three approaches to cognitive aspects of epilepsy are followed in the investigations to be reported here. Data were collected and analysed in order to discover whether or not in general cognitive ability the epileptic patients would show a normal distribution; whether or not any tendency to deteriorate in cognitive ability would appear on re-testing two years later; and whether or not, when compared with a control group, the epileptic patients would show any significant differences in such special mental abilities as memory, fluency, and vocabulary.

Study of 161 Epileptic Patients

Population.—One hundred and sixty-eight epileptic patients, children and young adults, attending an

out-patient clinic at the National Hospital, London, were tested individually between June, 1945, and December, 1949. In most cases the testing was done within a few weeks of the patient's first attendance at the clinic. This in large measure circumvented the selection problem mentioned by Somerfeld-Ziskind and Ziskind (1940) that patients attending hospital out-patient departments may not fairly represent the whole group of epileptics living outside institutions, for those who are not helped by the treatment tend to drop out. In all 207 test batteries were administered, 39 being repeat tests. Of the 168 different patients, seven were too young for adequate measurement with the battery of tests used, reducing the number in the group to 161. In 145 patients the medical diagnosis was " idiopathic " epilepsy ; in the remaining 16 the attacks were either known or suspected to have an organic basis. The attacks were of a minor character (petit mal) in 37 patients and either of a major (grand mal) or major and minor character in 124 patients.

Tests.—At the outset of the research it was known that the patients would vary greatly in age, so a battery of tests was designed which would enable an estimate of ability to be made from the age of 6 years up to adulthood. The following tests were used and after each are given the scores derived from them.

Cattell's (1936) *Test of Retentivity* (*Memory*).—Time allowed was one half minute for learning followed by one minute for recall for each of four parts : (1) page with 10 simple pictures ; (2) 10 nonsense syllables (to be recognized later among 20) ; (3) 10 geometric shapes (to be recognized later among 20) ; (4) 10 words read slowly twice to subject. Two scores were made : the total number of items given correctly at immediate recall ; the total number given at delayed recall after roughly one hour's testing.

Progressive Matrices (*Untimed Version*).—Board form of sets A and B were used for 8-year-olds and younger. Three scores were made : (1) the total number of problems correct converted into percentiles and thence into I.Q.s ; (2) the time taken ; and (3) " scatter " on separate sets.

Fluency Tests.—These consisted of the number of names of flowers given in one minute, the number of names of birds given in one minute, the number of different items under a tree (Fig. 48 in Cattell's (1936) book), and the number of different items in a street (Fig. 49 in Cattell's book). Two scores were made : (1) reproductive fluency, i.e., the number of flowers plus the number of birds, and (2) imaginative fluency, the number of items under the tree plus the number of items in the street.

Mill Hill Vocabulary Test.—Both A and B forms for definitions were used. One score, the number of correct definitions given, assessed in accordance with acceptable responses kindly supplied by Raven, was converted into verbal attainment quotients (V.A.Q.) from norms given by Raven.

Results.—The interest in this first investigation lies mainly with the general mental ability of epileptic patients, measured by intelligence tests, and not with their special abilities or disabilities, measured by memory and fluency tests. Accordingly, the summary of results in this section will be confined to the progressive matrices and Mill Hill vocabulary tests.

Raven's five grades on these tests are based on percentiles, where Grade I equals I.Q. 124 and over ; Grade II equals I.Q. 110 to 123 ; Grade III equals I.Q. 91 to 109 ; Grade IV equals I.Q. 77 to 90 ; and Grade V equals I.Q. 76 and under. Each patient's percentile score was calculated and converted into an I.Q. Out of the group of 161 patients, 20 were below I.Q. 70 on progressive matrices and the same number below V.A.Q. 70 on vocabulary. Therefore, of this fairly representative group of epileptic patients, over 12% were mentally defective as measured by these tests. For the whole group the median I.Q., as assessed by the progressive matrices was 94, and the median V.A.Q., as assessed by the Mill Hill vocabulary test, was 96.

Possibly of greater interest than these slightly under-normal medians for the epileptic group is the abnormal distribution of the matrices gradings. Table I shows the expected distribution of a normal population in the various grades and the distribution found for these 161 epileptics. The difference in distribution is statistically significant.

The group of epileptic patients has only a slightly below normal median I.Q., but the distribution of their gradings on the progressive matrices test differs significantly from a normal group. There are fewer patients with average scores (27·9% as compared with the expected 50%) ; a slightly

TABLE I

DISTRIBUTION OF EPILEPTIC PATIENTS' GRADING ON PROGRESSIVE MATRICES TEST COMPARED WITH NORMAL POPULATION

	Grade I	Grade II	Grade III	Grade IV	Grade V
Expected percentage of normal population	5	20	50	20	5
Percentage of epileptics	3·7	26·1	27·9	23·0	19·3

greater number above average (29·8% as compared with the expected 25%); and a great many more below average (42·3% as compared with 25%).

Intelligence quotients derived from the epileptic patients' scores on a non-verbal test, progressive matrices, and on a verbal test, Mill Hill vocabulary, are below the level of mental defect for over 12% of the group. This result confirms the findings of Sullivan and Gahagan (1935) who reported over 13% mentally defective in their group of 103 epileptic children.

Re-test Results

Population.—Thirty-nine children and young adults attending the Out-Patients' Clinic at the National Hospital, ranging in age from 5 to 19 years, with an average age of 11, were given re-tests. The group included 20 girls and 19 boys, eight having minor attacks, 20 major attacks, 11 both minor and major attacks; in one or possibly two patients the attacks had an organic origin, while in all the others they were considered to be idiopathic.

Tests.—These were the same as described in the previous paragraphs. The battery of tests was given individually to the patients and repeated approximately two years later (23·4 months on the average) under identical conditions and by the same psychologist. No well established norms exist for the memory and fluency tests, so it is impossible to ascertain whether or not improvement, if any, on these tests is in line with normal expectations. Comparison results on test and re-test will be limited to the Progressive Matrices and Mill Hill Vocabulary Tests.

Results.—These were analysed under two headings, whole group and medical categories.

Whole Group.—The percentile scores on the progressive matrices were transformed into I.Q. terms. On the second testing, as is to be expected, the average I.Q. is slightly higher than on the first testing: the average on first testing was 97·2 and on the second testing 99·2. There was very great individual variation from first testing to second: 22 patients varied more than 5 points of I.Q.; 10 more than 10 points. Using Raven's suggested grades, it was found that the correlation between grade on first testing and grade on second testing was 0·76.

Using the Mill Hill Vocabulary Test Raven's suggested working of the V.A.Q. as

$$\frac{\text{verbal mental age}}{\text{chronological age}}$$

was followed. The distribution curve is considerably flattened, at least for this particular group. The average V.A.Q. on first testing was 99·2, and on second testing 95·4. It would be anticipated that the average V.A.Q. would be at least as high on the second testing if the vocabulary of the epileptics were growing at the normal rate. This considerable drop may find some partial explanation when the results are analysed further as below.

The V.A.Q.s were converted into grades for comparison with progressive matrices results. As with the matrices there is a great deal of individual variation in gradings on the two testings. The distribution curve is much flatter than for matrices so it might be expected that many more individuals would change from one grade to another: however, 18 patients were unchanged, 13 were one grade down, six were one grade up, and two were two grades up. The correlation between grade on first testing and grade on second testing is 0·82.

TABLE II

RESULTS ON FIRST AND SECOND TESTING FOR PROGRESSIVE MATRICES AND MILL HILL VOCABULARY TESTS

	Average I.Q. 1st Testing	Average I.Q. 2nd Testing	Average V.A.Q. 1st Testing	Average V.A.Q. 2nd Testing
18 " improving "	98·8	100·1	106·7	105·6
19 " unchanged " or " worsening " ..	97·6	100·3	94·1	88·4

Medical Categories.—With regard to their clinical progress, 18 were considered to be improving, that is having fewer attacks, 16 unchanged, and three were somewhat worse (two had left the clinic). As the " worsening " group was so small it was decided to put the " unchanged " and " worsening " groups together for comparison with the " improving " group. The comparative results on the first and second testings for the Progressive Matrices Test and Mill Hill Vocabulary Test are shown in Table II.

On the Progressive Matrices Test there is little difference between the two groups in average I.Q. at first testing, but the " improving " group on second testing show somewhat less improvement than the " unchanged " or " worsening " group. Neither difference is significant. On the Mill Hill Vocabulary Test the " unchanged " or " worsening " group is initially well below the " improving " group, and there is only a slight fall in the " improving " group at the second testing while the other

group is significantly lower on the second testing than on the first. The two subjects who showed an increase of two grades in the vocabulary test were both " improving " cases.

It has sometimes been suggested that different types of epileptic attacks are correlated with different degrees of deterioration. The data from this investigation were analysed to discover whether or not they would show a differential change for the various types, and a summary is given in Table III.

TABLE III

RESULTS ON FIRST AND SECOND TESTING FOR PROGRESSIVE MATRICES AND MILL HILL VOCABULARY TESTS ACCORDING TO DIFFERENT TYPES OF ATTACKS

No. of Cases and Type of Attack	Average I.Q. 1st Testing	Average I.Q. 2nd Testing	Average V.A.Q. 1st Testing	Average V.A.Q. 2nd Testing
8 with minor	99·0	97·9	112·5	103·5
20 with major	96·1	98·8	96·7	92·7
11 with major and minor	97·9	100·8	94·4	94·7

TABLE IV

RESULTS ON FIRST AND SECOND TESTING FOR PROGRESSIVE MATRICES AND MILL HILL VOCABULARY TESTS OF PATIENTS WITH NORMAL AND INTERRUPTED EDUCATIONS

No. of Patients	Average I.Q. 1st Testing	Average I.Q. 2nd Testing	Average V.A.Q. 1st Testing	Average V.A.Q. 2nd Testing
6 patients with interrupted education..	93·8	89·5	92·3	85·7
24 patients with normal education ..	103·0	104·5	108·2	106·8

On the progressive matrices none of the differences between first and second testings is statistically significant. On the Mill Hill Vocabulary Test the whole group shows a tendency to a lower V.A.Q. on re-testing, but when the results are subdivided according to kind of epilepsy it is apparent that it is those with minor attacks who show this most markedly. This difference, however, does not quite reach the level of statistical significance.

For comparison with the results given in Table II it might be noted that of the eight patients with minor attacks six are reported to be " improving ", one has left the clinic, and one has shown no change.

Educational Facilities.—Of the group of 39, two were no longer attending the clinic and information as to their education was not available. Only eight of the remaining 37 had their education interrupted due to their attacks. Two of these were past school age when the second testing was done, and so the previous interruption in their education could have no bearing on the difference between their vocabulary rating at the two testings. Of the 29 patients known to have had no interruption in their education, five were over school age at the time of their second testing, leaving 24 cases where education was proceeding normally during the interval between testings and six where it was interrupted to some extent. In Table IV are set out the average quotients on the two main tests for the first and second testing for the two groups.

At the first testing the two groups show large average differences in both verbal and non-verbal intelligence. At the second testing those whose education has been irregular drop over 4 points in I.Q. on progressive matrices, but the difference is not significant. On the vocabulary test those whose education has been interrupted drop nearly 7 points in V.A.Q. and this difference is statistically significant.

The correlation between grade on the progressive matrices for the first and second testing is 0·76. The reliability of this test is usually found to be somewhat lower than that of verbal intelligence tests, namely about 0·85. It is to be expected that with an interval of two years between the testings the correlation will be rather lower. Keir (1949) reported a correlation of 0·64 between successive trials by a small group of 11-year-old children with an interval of two years between testings. The author (1945), using only the board form of the test on a group of 84 senile dementia patients, reported a reliability coefficient of 0·49 when the second trial was made four months after the first. It would appear that the correlation of 0·76 between successive trials by the epileptic patients with an interval of two years is not abnormal.

The correlation between grade on the Mill Hill Vocabulary Test for first and second testing was found to be 0·82. This would seem to be rather low for this test even with an interval of two years. For

comparison it is interesting to note that the author (1945) reported a reliability coefficient of 0·96 for this test when given to senile dementia patients with an interval of four months. The unusually low correlation found for the epileptic patients suggested that it might be worthwhile analysing the results in greater detail.

When the results of test and re-test are studied separately for the patients reported as " improving " and those reported as " unchanged " or " worsening ", it is found that the drop in vocabulary quotient for the " unchanged " and " worsening " group is statistically significant. Taking the patients with each type of epileptic attack separately, it is found that none of the differences reaches the level of statistical significance. The group suffering from minor attacks shows the largest drop in vocabulary. (The probability of this difference being due to chance is 1 in 10.) Lennox (1944) reported that " cases with pure petit mal may go unscathed " as regards deterioration. The number of patients of this type in this investigation is too small for the suggestive deterioration in vocabulary rating to be regarded as a serious refutation of his findings, but it would be interesting to know what further research could discover in this matter of differential deterioration of the various epileptic types.

The patients whose education has been interrupted show a significant fall in vocabulary quotient. This result would seem to suggest that being kept from school because of their attacks has meant that there has been a serious retardation in the normal rate of vocabulary growth.

None of the differences found between test and re-test on progressive matrices is statistically significant.

Comparison of 57 Epileptic Children with a Control Group

Population.—Fifty-seven epileptic children were investigated, this being the total group of 7- to 14-year-old children diagnosed as suffering from idiopathic epilepsy with an I.Q. 75 or over on the progressive matrices who were given all the tests.

Tests.—The battery of tests as described previously had been given to the 57 epileptic children, and the same tests were given to the control group*, matched one to one for sex, for age within three months, and as nearly as possible for score on the progressive matrices.

Results.—The chief purpose of this investigation was to discover whether or not any differences would be found between epileptic and normal children, matched for age, sex, and non-verbal intelligence, in other mental abilities, as measured by the memory tests, fluency tests, and vocabulary.

The average age of the epileptic group and the normal group was identical, but despite time and care in matching the matrices score the control group had nearly one point lower score on the average, which fact should make somewhat more significant any superiority shown by the controls over the epileptics on the other tests.

The scatter was calculated for each child's score on the progressive matrices, Raven's suggestion being that a high scatter is indicative of a rather lower reliability. (" Scatter " is the sum of the differences between the child's score on each of the five separate sets and the median scores made by subjects whose total score on the whole test is identical.) The average scatter for the controls was 3·5 and for the epileptics 3·0, indicating that the epileptics' scores were possibly slightly more reliable. This difference does not reach the level of statistical significance. A much larger difference appears on the time taken to complete this test : the average time spent by the controls was 16·7 minutes and by the epileptics 21·3 minutes. This difference is statistically significant but too much stress cannot be placed on it, for it is undoubtedly possible that the time taken on an individual test of this type is as much due to the tester as to the child.

With regard to the results of the test of retentivity (memory), the group of 57 control children recalled on the average 24·9 items immediately after learning and the epileptic children 23·8 items. This difference is not significant. When asked again at the end of the testing session to recall the items the controls produced 18·6 items and the epileptics 16·0. This difference also fails to reach the level of statistical significance.

With regard to the fluency test results, the pattern is reversed. The total scores on the two tests of naming flowers and birds were taken together as " reproductive fluency " and those on the two tests of giving possible items under the tree and in the street together as " imaginative fluency ". Here the epileptics showed a superiority : for " reproductive fluency " their average was 16·3 and the controls' average was 15·1 ; for " imaginative fluency " the epileptics' average was 18·1 and the controls' was 14·0. The difference in " reproductive fluency " is not significant, but the difference in " imaginative fluency " is statistically significant.

Again, in the scores on the vocabulary test the epileptic group showed a small superiority. Their average score was 27·0 and the controls' average was 25·7. This difference is not statistically significant.

* Miss S. Rostal was good enough to do the matching and testing of the control group.

Somerfeld-Ziskind and Ziskind (1940) found that epileptics are defective in memory retention, and this would seem to be a widely accepted belief. In the present investigation the difference in immediate memory between the epileptic children and the control group is below the level of statistical significance, and the difference in delayed recall, while somewhat larger, fails to reach the level of significance. (The probability of its being due to chance is 1 in 10.) These results, therefore, would seem to lend little support to the view that epileptics are particularly poor in memory. It is also to be remembered that all the epileptic children were being treated with anti-convulsants.

Capps (1939) and also Somerfeld-Ziskind and Ziskind (1940) mention a general defect in language ability among epileptics, but the results reported here do not bear this out. In the fluency tests and in the vocabulary test the advantage lies with the epileptics, but the only difference to reach the level of statistical significance is the one in " imaginative fluency ". The explanation of this superiority on the part of the epileptics in these language tests may be twofold : the epileptics' very small advantage in non-verbal intelligence, and the probably slightly better social and cultural background of the epileptic group compared to the controls who lived in a crowded south London borough. In addition it is not known what the effect on the results may be from having two different testers, though care was taken to try to ensure that identical instructions and encouragement were given, and the author scored the responses of both groups.

Summary and Conclusions

The average intelligence of a group of 161 epileptic patients is only slightly below normal : median I.Q. is 94 on the Progressive Matrices Test and the median V.A.Q. is 96 on the Mill Hill Vocabulary Test.

The distribution of gradings on the Progressive Matrices Test of the epileptic patients differs significantly from that of a normal population.

Over 12% of the 161 epileptic patients are below the level of mental deficiency on both the verbal and non-verbal test.

A group of 19 patients whose condition is considered " unchanged " or " worse " show a significant fall in vocabulary quotient on re-testing after an interval of two years : 18 patients whose attacks are decreasing show no significant fall.

Epileptic patients with minor attacks show a larger fall in vocabulary quotient on re-testing than those with major or major and minor attacks but none of the differences is significant.

Patients whose education has been interrupted due to epilepsy show a significant fall in vocabulary quotient on re-testing.

No group of epileptic patients shows a significant change in progressive matrices quotient on re-testing.

A group of 57 epileptic children, matched for sex, age, and score on progressive matrices score with a control group, shows a lower scatter of scores on separate sets of progressive matrices. This difference is suggestive of somewhat greater reliability but is not statistically significant.

The epileptic children took longer to do the Progressive Matrices Test than the control group. This difference is statistically significant but it is possible that part of the difference is due to the fact that two different testers administered the tests.

The epileptic children's inferiority in immediate memory is not significant, and their inferiority in delayed recall, while somewhat greater, is below the level of statistical significance.

The epileptic children are superior in both fluency scores : in " reproductive fluency " the difference is not significant but in " imaginative fluency " it is statistically significant.

The epileptic children show no significant difference in vocabulary.

The London County Council was kind enough to arrange facilities for testing a control group of normal children in three schools, and my thanks are due to the Education Officer of the L.C.C. and to the head teachers of the Oliver Goldsmith Junior Mixed School, Albany Road Secondary Girls' School, and Southampton Way Secondary Boys' School for their courtesy and helpfulness.

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HERPES ZOSTER OPHTHALMICUS AND POST-HERPETIC NEURALGIA

BY

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Jonathan Hutchinson (1865–6) was the first person to distinguish herpes zoster ophthalmicus from erysipelas, and he described 18 cases, one of which developed a third nerve palsy. Hutchinson stated that zoster never affected the cheek at all, but a few years later Head and Campbell (1900) described a case with affection of the mandibular branch; Paton (1926) reported a case with involvement of both cheek and forehead; and O'Neill (1945) described what he considered to be the first case with involvement of the first, second, and third divisions of the trigeminal nerve, but in his case there was also involvement of the seventh and eighth cranial nerves.

Various palsies of the extraocular nerves may occur, and the incidence according to Worster-Drought (1923) is 7% of cases with ophthalmic herpes zoster. The commonest nerve to be paralysed according to Moore (1932) is the third, and forms 74% of the extraocular nerve pareses; Edgerton (1942) states that the third nerve was the commonest extraocular nerve to be paralysed and occurred 96 times in the literature. Edgerton (1942), in his review of the literature, stated that paralysis was found usually after, sometimes with, and occasionally before, the skin eruption; he goes on to comment that paralysis of the extraocular nerves is due to "encroachment of the inflammation in the region of the cavernous sinus": this is probably incorrect as the lesion has been shown to be in the anterior horn cells when muscular paralysis occurs elsewhere, so there is no reason to postulate a different pathology so far as the eye palsies are concerned. Other cases of herpes zoster ophthalmicus with intraocular or extraocular muscular palsies have been described by Parkes-Weber (1916), Hall (1903), and Mules (1903). Mules also suggested that ocular palsies could occur without an eruption of zoster, i.e. *formes frustes*.

Batignani (1934) reported a case of moderate exophthalmos with total ophthalmoplegia; Carmody (1937) described severe exophthalmos in the course of herpes zoster ophthalmicus and more recently Donahue (1946) described a case of ophthalmic herpes zoster with the development of complete external ophthalmoplegia occurring during the eighth week of the disease.

Achard and Castaigne (1897) described a permanent paralysis with dilation of the left pupil after an attack of ophthalmic zoster, and Parkes-Weber (1916) suggested, as had Mules, that unexplained mydriasis and attacks of paresis in isolated muscles or muscle groups, with or without sensory loss, might be "herpes zoster sine herpete".

The age of onset of ophthalmic herpes zoster in 2,250 cases reviewed by Edgerton (1942) was 44, with a sex incidence of 54% males: the same author also recorded a low incidence of herpes zoster ophthalmicus, there being five cases out of 30,000 patients at an eye hospital during one year.

Pathology

Von Barensprung (1863) was the first to perform a post-mortem examination on a case of zoster and found a lesion in the posterior root ganglion. Sattler (1875) reported a case of herpes zoster ophthalmicus where a necropsy was performed 14 days after the onset; the Gasserian ganglion was found to be infiltrated with small, round cells and there was destruction of the ganglion cells. Head and Campbell (1900) investigated very fully the pathological changes at the posterior root ganglia, and Denny-Brown, Adams, and Fitzgerald (1944), summarizing the pathology, state: "The present concept of herpes zoster is an acute inflammatory reaction in isolated spinal or cranial sensory ganglia, posterior grey matter of cord or adjacent leptomeninges". The lesion in their cases resembled an infarct necrosis with ganglionitis marked by necrosis of all or part of the ganglion with or without haemorrhage; in addition there was poliomyelitis with a mild leptomeningitis localized to affected

segments, and lastly a true peripheral mononeuritis.

Brown and Dujardin (1919) investigated the cerebrospinal fluid in herpes zoster; they found that there was a variable lymphocytic response, but that it bore no relation to the intensity of eruption, to age, or to the stage of disease, and varied from normal to 470 lymphocytes per c.mm.

Vascular Supply of the Gasserian Ganglion

Bergmann (1942) quotes Meckel (1748) as making the first observation on the arterial supply of the trigeminal ganglion. Bergmann found that there was most constantly an arch on the medial side of the ganglion around the emerging primary divisions: the convexity of this arch is forwards from a small branch off the internal carotid artery, the lower part from the middle mengingeal or small meningeal arteries, and also from a branch of the internal maxillary artery through the foramen rotundum and occasionally through the foramen lacerum.

Truex (1940) investigated changes which occurred with age in the Gasserian ganglion. He examined ganglia from necropsies with age variations from 28 months to 66 years, and he found that fatty degeneration of sensory ganglion cells appeared to be the cause of loss of many neurons after middle age. He suggested that it might cause decreased sensitivity after the third decade. He also concluded that elastic intimal thickening in axial arteries suggested that vascular disturbances might be a factor in these degenerative processes. These factors may explain the greater incidence of post-herpetic pain in older people.

Present Investigation

Fifty-eight unselected patients who had had herpes zoster ophthalmicus and had attended Moorfields Eye Hospital, London, from 1941 to 1948 were investigated with special reference to the incidence of post-herpetic pain.

The average age of onset was 53·7 years in 57 of the 58 patients, (the age of one patient, who was later untraced was not recorded), and the ages varied from 19 to 85. There was no significant difference in the sex incidence: 27 females and 31 males.

Of the 58 patients, no case showed any affection other than on the first division alone, and, although the series is a small one, this collaborates the rarity of affection of the other divisions as reported by other authors. The right side was affected in 33 cases and the left in 24 cases, the side in an untraced case being unrecorded.

The 58 patients were sent a postal questionnaire asking about the presence of pain on the area involved with the shingles and also the presence of any " numbness ". Forty-seven patients answered, the remaining 11 being untraced. Only one patient had had such severe pain as to necessitate any radical treatment, an alcoholic injection in the Gasserian ganglion; unfortunately this patient could not be examined as he had died: no other patient had had deep *x*-ray treatment, leucotomy, or prolonged medical treatment. Three other patients had also died before follow-up, but, according to relatives, had had no persistent pain after the herpes. Forty-three patients who were living answered the questionnaire; 14 of these were not examined, and their history should, therefore, not be accepted in great detail, but two patients with " numbness " reported that they had " pain when tired ", one patient had numbness but no pain, and the rest had neither numbness nor pain.

Twenty-nine patients were examined; all the patients had had pain in the face and in the eye during the period of vesicle formation and for some weeks before the lesions healed, but 13 patients (aged 51, 34, 71, 51, 60, 36, 19, 61, 51, 33, 66, 41, and 54 at the onset of the herpes) had had no pain or irritation whatsoever afterwards and, excluding corneal scarring and optic pallor, had no pupillary changes or any sensory loss at all except where skin scarring was severe. Eight patients, with no abnormal painful sensations whatsoever, had varying pupillary and sensory abnormalities which are discussed later. As regards the incidence of pain, one patient with no sensory loss or pupillary abnormality had a " dull aching sort of pain in the forehead since ", which was worse with worry or fatigue. Seven other patients had painful sensations varying in degree from a " pricking irritation " to " sharp, stabbing, neuralgic pains ". In all these patients there was some form of sensory loss corresponding to the anatomical distribution of the first division of the fifth nerve.

Case Reports

Case 1.—A woman, aged 49, had herpes zoster affecting the left ophthalmic division in 1948. She was still having occasional " pin pricks " and frequent " itching " in the forehead. She was found to have blunting of wool, pain, and temperature sensation in the distribution of the first division of the fifth nerve. She described herself as a nervous, worrying person, but had never had any nervous breakdowns.

Case 2.—A man, aged 39, had herpes zoster affecting the right ophthalmic division in 1948. He was still having occasional " pricking " sensations in the forehead. He was found to have blunting of all sensory modalities in the distribution of the first division of the fifth nerve. He described himself as a nervous, worrying type, and had been discharged from the R.A.F. on account of neurosis.

DETAILS OF CASES IN PRESENT SERIES

	Total	Ages (in years)								
		<20	<30	<40	<50	<60	<70	<80	<90	Unrecorded
Total	58	1	1	7	14	15	16	2	1	1
Total traced (including those dead)	47	1	1	6	11	12	13	2	—	1
Total examined	29	1	—	5	9	8	5	1	—	—
Sensory loss	12(2)	—	—	2	6(1)	3	1(1)	—	—	—
Sensory loss with painful sensations	7(2)	—	—	2	3(1)	1	1(1)	—	—	—
Pain without sensory loss	1*	—	—	—	—	1	—	—	—	—
Percentage of painful sensations in patients examined	—	—	—	—	40%	33%	12·5%	20%	—	—
No. with pupillary abnormalities..	6	—	—	1	—	3	2	—	—	—

Figures in parentheses indicate patients not examined. *Denotes pain described as "dull aching".

Case 3.—A woman, aged 50, had herpes zoster affecting the right ophthalmic division in 1946. She still had quite severe post-herpetic pain, which she described as a "severe toothache like a nerve exposed", in the forehead. She was found to have considerable blunting of pain and touch sensation; temperature sense was abnormal, heat producing a prickling sensation and cold a hyperpathia in the distribution of the first division of the fifth nerve. She described herself as a normal person, and not subject to any nervous disability: she had been separated from her husband for some years and had been at work for 24 years.

Case 4.—A woman, aged 41, had herpes zoster affecting the right ophthalmic division in 1944. She still had an occasional "pricking sensation", but it was only felt in the scarred area of the skin. She was found to have very severe skin scarring, and the only sensory loss (which was to all modalities) was in this area. She described herself as a nervous person and had had a "nervous breakdown" in the blitz.

Case 5.—A man, aged 34, had herpes zoster affecting the left ophthalmic division in 1942. He was still having an occasional "burning irritation" in the forehead, but only when he was worried or anxious. He was found to have abnormal skin sensation: testing with pain produced a burning sensation, and there was also a hyperpathic response to touch and temperature of the first division of the fifth nerve.

Case 6.—A woman, aged 46, had herpes zoster affecting the right ophthalmic division in 1948. She was still having occasional smarting, burning, "neuralgic" pains in the forehead. She was found to have diminution of touch and pain sensibility over the distribution of the first division of the fifth nerve. She described herself as a very nervous person, and had had a nervous breakdown in the past.

Case 7.—A woman, aged 61, had herpes zoster affecting the left ophthalmic division in 1941. She was still having occasional itching and tingling in the forehead. She was found to have reduction of pain, temperature, and touch sensation over the first division of the fifth nerve. She described herself as a normal person and was not a worrying, anxious type.

From the histories of these patients there appears to be a distinct correlation between sensory loss and abnormal sensation. One patient, previously described, had what she called an "ache" in the forehead, but no sensory loss at all: all the other seven patients with pain or abnormal sensation had definite sensory loss or hyperpathia. In addition, five other patients had sensory loss over the first division of the fifth nerve which had been affected by herpes. These five patients described themselves as normal persons who were not worriers and had never had any nervous breakdowns. Although the number of patients in this series is small the impression left is that it is the worrying, anxious type with sensory loss who may develop abnormal pain sensations.

Six of the 29 patients examined had abnormal pupillary responses which could not be accounted for by corneal scarring or blindness. The abnormal pupils, occurring on the same side as the herpes zoster, showed no reaction to direct or consensual light at all, but responded well on accommodation. They could not be regarded as true Argyll Robertson pupils, as they were not always small, were not irregular, and showed no changes in pigmentation of the iris, but the high percentage of patients showing such an abnormality was surprising.

Discussion

The aetiology of post-herpetic neuralgia is unknown, but it has been suggested that it may be assc-

ciated with vascular changes due to arteriosclerosis, as it is more frequent in elderly people. In this small series there was no apparent correlation with age at all, and this suggests that vascular changes are not a definite factor. The incidence of post-herpetic neuralgia appears small ; only one patient had had severe neuralgia requiring injection (although most authorities agree that this has no therapeutic effect anyway), and seven others had uncomfortable paraesthesiae. All the patients who were examined and had these paraesthesiae had a marked sensory loss corresponding to the distribution of the sensory symptoms, but there must be some other aetiological factors involved as five other patients had sensory loss, but no paraesthesiae. Psychogenic factors have an important bearing on the subject of the pain, and this has been corroborated by the results of frontal leucotomy. In this investigation there was an impression that the patient who continued to have pain after herpes zoster was of the introspective, worrying type, but always in association with sensory loss : the pain was, in fact, always made worse by worry and tiredness. The pain appears gradually to improve as time passes, but can continue for a number of years : it is never delayed in onset, always being present from the start of the vesicle formation.

The pupillary abnormalities in these cases did not appear to be associated necessarily with either pain or sensory loss, three of the patients having no sensory abnormalities at all, nor could these abnormalities be correlated in any way with the severity of the attack of herpes. Pseudo Argyll-Robertson pupils have been reported in herpes zoster ophthalmicus by Guillain, Lagrange, and Perisson (1926) and Valière-Valeix (1931). Nathan and Aldren Turner (1941) described pseudo Argyll-Robertson pupils in cases of traumatic lesions to the peripheral efferent pathway. They suggested that accommodation-convergence fibres might by-pass the ciliary ganglion and go direct to the episcleral ganglia described by Axenfeld (1907) and Givner (1939), and that this might explain the presence of accommodation-convergence responses and the absence of light reflexes in such traumatic lesions. Their arguments are not necessarily valid because their cases had had third nerve palsies, and such pseudo Argyll-Robertson pupils may be due to abnormal regeneration of the oculo-motor nerve, as has been reported by Ford, Walsh, and King (1941), by Bender (1945), and by Ritchie Russell and Wright (1948). In the cases reported in this series no third nerve palsy had been observed previously, and therefore synkinetic pupillary phenomena due to a recurring third nerve palsy could be excluded. The exact anatomical site of the lesion is doubtful in the absence of post-mortem material, but in the absence of gross change in visual acuity and of a consensual light response, the site of the lesion to account for these pseudo Argyll-Robertson pupils could be in the ciliary ganglion.

Summary

Fifty-eight patients who had had herpes zoster ophthalmicus were investigated : 47 were traced, and in only one patient had alcoholic injection been given for severe pain.

Twenty-nine patients were examined, and of these seven had persistent pain or abnormal sensations.

The seven patients with pain or abnormal sensations were all found to have diminution of some sensory modalities.

Five other patients examined also had sensory diminution, but did not have any pain or other abnormal sensation.

Six out of the 29 patients examined had pupils which did not react at all to direct or consensual light, but reacted well to accommodation.

I am grateful to the consultant surgeons of Moorfields Eye Hospital for permission to follow-up and examine their cases, and especially to Mr. Robert Davenport, Senior Surgeon, for arranging for me to do so.

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Third Symposium Neuroradiologicum

The third Symposium Neuroradiologicum will be held in Stockholm on September 17–20, 1952. The president is Professor Erik Lindgren, Stockholm. All those wishing to attend should send in their names and addresses to the Secretary, Symposium Neuroradiologicum, Serafimerlasarettet, Stockholm, Sweden.

International Meeting of Neuropathologists

The first international meeting of neuropathologists will be held in Rome from Monday, September 8, to Saturday, September 13, 1952. The President is Dr. Mario Gozzano (Rome), and the Secretary General, Dr. Armando Ferraro (New York).

Final arrangements are now being made for the programme. It has been decided that the following subjects should be discussed, one day being devoted to each subject: (1) histopathology of the demyelinating diseases; (2) histopathology of vascular diseases; (3) histopathology of schizophrenia; (4) histopathology of mental deficiencies; and (5) histopathology of senility.

Also part of one or more days will be devoted to short papers of special interest not related to the above subjects and to cinematographic and microscopical demonstrations.

The British Committee will be glad to receive offers of short papers for this meeting, especially of papers related to the five main topics. Those who wish to read papers should communicate with Dr. J. G. Greenfield at the National Hospital, Queen Square, London, W.C.1, or with any member of the British Committee, giving the full title of the paper and a summary showing in what respects it covers new ground.

British Committee: Dr. J. G. Greenfield, Hon. President; Prof. Dorothy S. Russell, Vice-President; Prof. J. H. Biggart; Prof. A. Meyer; Dr. W. Blackwood; Dr. W. H. McMenemey; Dr. R. M. Norman.

J. Neurol. Neurosurg. Psychiat., 1952, **15**, 50.

THE EFFECT OF DENERVATION ON NAIL GROWTH*

BY

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Weir Mitchell (1872) and Sharpey-Schafer (1931) reported that section of a nerve supplying cutaneous fibres to the fingers results in a retardation of nail growth. Head and Sherren (1905), however, believed that the slowing of growth observed in these cases was due entirely to the immobilization of the limb either as a result of splinting or paralysis and that division of sensory fibres without limitation of digital movements is not associated with any deficiency in nail growth. According to Bernhardt (1881) " nail growth is relatively independent of the influence of the nerve fibres innervating the fingers and tissues of the nail bed ", though his method of measuring growth from " the upper margin of the epidermis covering the nail root " was open to error. In view of these few and contradictory observations it was decided to investigate nail growth in a series of patients with peripheral nerve injuries.

Material and Methods

Observations on the effects of denervation on nail growth were made on 10 patients with peripheral nerve injuries (Table I). In three of these the ulnar nerve and in six the median nerve had been severed and repaired. One patient sustained a crush injury of the right median nerve when it was caught between the jaws of a Spencer Wells forceps applied to a lacerated brachial artery in a wound in the upper arm. This resulted in completely interrupting conduction in the fibres innervating the digits for more than six months. In five of the median nerve injuries there was a concomitant injury of the brachial artery which necessitated ligation of that vessel. This introduced a complicating vascular factor at the periphery of the limb which, despite the establishment of an adequate collateral circulation, may have contributed to deviations from the normal rate of growth which were observed in these cases.

For data relating to the rate of nail growth in normal digits under a variety of conditions and to the range of normal variation in that growth we have relied on the valuable paper by Le Gros Clark and Buxton (1938). Our technique for measuring nail growth was substantially the same as that advocated and used by them except that we did not attempt a degree of accuracy in measurement greater than 0·25 of a millimetre which we found suitable for the purposes of our enquiry. Readings in most cases were made at weekly intervals. As one mark on the nail advanced distally, another was made at a specified distance from the margin of the lunula. Thus the number of recordings registered in column 4 of Table I refers to the number of marks which were made and traced distally.

The difficulty of obtaining suitable clinical material for prolonged and unbroken observations explains the small numbers of cases available for the enquiry. However, these have been investigated in some detail and cover all seasons of the year.

Observations were made on the nails of the thumb, index, and little fingers. The index and little fingers provide the most dependable readings because the nail beds of these digits are innervated solely by the median (index) and ulnar (little) nerves. The ulnar nerve distribution in the hand commonly includes part or all of the ring finger and may extend to involve the middle finger while the nail bed of the thumb is innervated by the superficial radial nerve as well as by the median. For these reasons we attach importance only to the data provided by measurements of nail growth of the index and little fingers following lesions of the median and ulnar nerves respectively.

Results

Relevant data relating to the nerve injury sustained by the patients investigated are given in Table I. Details relating to the growth of the nails in the denervated and non-denervated digits are given in Tables I and II.

Discussion

An analysis of the data recorded justifies the following statements.

* This work was assisted by a grant from the National Health and Medical Research Council of Australia.

TABLE I

RATE OF GROWTH IN DENERVATED AND NON-DENERVATED DIGITS

Patient	Nerve Involved and Nature of Lesion	Arterial Ligation	Marks Traced	Time (days) and Distance* (mm.) Grown and Average Rate of Growth (μ/day)											
				Thumb				Index				Little			
				R	L	Average		R	L	Average		R	L	Average	
						R	L			R	L			R	L
M.I.T.	Left ulnar suture mid-forearm	No	1					4/56	3/56	71	54	3/56	3/56	54	54
N.D.S.	Right ulnar suture mid-upper arm	No	1					4/36	4.5/36	111	125	4/36	4/36	111	111
			2					6/57	6.5/57	105	114	5/57	6/57	88	105
			3					5/42	5/42	119	119	4.5/42	4.5/42	107	107
J.M.	Left ulnar suture upper forearm	No	1	8.25/97	8.5/97	85	88	9/97	8.75/97	93	90	8.75/97	7.5/97	90	71
			2	5.5/68	7.0/68	81	103	6.3/68	6.5/68	93	96	8.0/68	6.0/68	118	88
P.L.	Left median suture mid-forearm	No	1					4/42	3.5/42	95	83				
			2					2.5/29	3/29	86	103				
			3					5/56	4/56	89	71				
			4					6/56	4.5/56	107	80				
J.R.L.	Right median suture elbow	Yes	1					5/79	7/79	63	89				
			2					4/44	5/44	91	114				
			3					4/40	5/40	100	125				
			4					5/51	7/51	98	137				
			5					5/54	6.5/54	93	120				
			6					6/70	7.5/70	86	107				
B.P.	Left median suture upper arm	Yes	1	5.5/72	4/72	76	55	5/72	3/72	69	42	3.5/60	3.0/60	58	50
			2	7/87	6/87	80	69	4/42	3.25/42	95	77	4.25/54	4/54	79	74
			3	3.25/35	2/35	93	57	3.25/35	1.5/35	93	43	2.5/35	2.5/35	71	71
B.S.	Right median suture upper arm	Yes	1	3.5/36	4/36	97	111	3.5/36	4/36	97	111				
			2	3/37	4/37	81	108	3.5/37	4/37	95	108	4.5/28	3.5/28	161	125
W.R.	Right median suture upper arm	Yes	1	3.5/42	5.5/42	83	131	3/42	5/42	71	119	3/42	4.5/42	71	107
			2	5/45	5.5/45	111	122	5/45	6/45	111	133	5/45	5.5/45	111	122
			3	5.25/48	6/48	109	125	5/48	6/48	104	125	5.5/48	5.5/48	115	115
E.M.G.	Right median cross anastomosis of superficial radial to median in the forearm	No	1	3.5/49	5/49	71	102	2/49	5/49	41	102	4.5/49	4/49	92	82
L.H.	Right median crush upper arm	Yes	1	4.0/52	6.0/52	77	115	3.5/45	>5.0/45	78	111	5/52	6/52	96	115
	Spontaneous recovery		2	5/54	6/54	93	111	4.5/54	>6.5/54	83	120	5.5/54	5.5/54	102	102
			3	4.5/47	5/47	96	106	3.5/47	6/47	74	128	4.25/47	4.25/47	90	90
			4	5.5/63	7.5/63	87	119	4.5/63	5.75/63	71	91	6.25/63	>6.25/63	99	>99
			5	6.3/62	7.5/62	100	121	5.5/62	7.5/62	89	121	7.0/62	6.5/62	113	105
			6	6.5/66	7.0/66	98	106	6.0/66	>7.0/66	91	>106	6.0/66	7.0/66	91	>106

*The value for distance precedes that for time.

TABLE II

AVERAGE RATE OF GROWTH FOR FULL PERIOD OF EACH INVESTIGATION

Patient	Nerve Involved and Nature of Lesion	Arterial Ligation	Number of Marks Traced	Average Rate of Growth (μ/day)					
				Thumb		Index		Little	
				R	L	R	L	R	L
M.I.T.	Left ulnar suture ..	No	1	—	—	71	54	54	54
W.D.S.	Right ulnar suture ..	No	3	—	—	112	119	102	108
J.M.	Left ulnar suture	No	2	83	95	93	93	104	82
P.L.	Left median suture ..	No	4	—	—	86	80	—	—
J.R.L.	Right median suture ..	Yes	6	—	—	88	115	—	—
B.P.	Left median suture ..	Yes	3	83	60	86	47	69	65
B.S.	Right median suture ..	Yes	2	87	109	96	109	161	125
W.R.	Right median suture ..	Yes	3	101	126	95	126	99	108
E.M.G.	Right median cross anastomosis of superficial radial to median ..	No	1	71	102	41	102	92	82
L.H.	Right median crush, spontaneous recovery ..	Yes	6	92	113	81	113	98	103

Rate of Growth in Nerve Lesions Unassociated with Ligation of the Main Artery to the Limb.—Uncomplicated denervation of the digits had a variable effect on nail growth. The denervation was without effect in one patient, a questionable slowing was recorded in two, and in a fourth growth was slightly but definitely retarded. In another patient, who had sustained an irreparable injury

of the median nerve, the affected hand was the seat of articular and periarticular changes and trophic disturbances. In this case there was a most pronounced slowing of nail growth in the thumb and index finger while growth in the little finger proceeded normally.

It is unlikely that the retardation recorded could be attributed to immobilization. The affected parts were not splinted and were subjected to frequent passive movements throughout the investigation. Furthermore nail growth in the non-denervated digits of the involved hand was not affected. Finally, patients with similar lesions and treated in precisely the same way presented individual differences in which growth was slowed in some and not in others.

Rate of Growth in Median Nerve Lesions Associated with Ligation of the Brachial Artery.—In contrast to the occasional and slight slowing recorded after uncomplicated nerve injury, a pronounced retardation of nail growth was observed in the index finger and thumb in every patient who had sustained a combined neurovascular injury. With one exception the index finger was affected to a greater degree than the thumb.

In such complicated nerve injuries nail growth was slightly slowed in the non-denervated digits in the initial stages when the circulation of the peripheral parts of the limb was affected. As the circulation improved, however, this slowing was corrected and the rate then equalled that on the opposite side (Fig. 1).

It is noteworthy that the co-existence of a vascular and nerve injury was consistently associated with a retardation of nail growth which greatly exceeded that observed after the uncomplicated nerve injuries. It is difficult to determine to what extent this disturbance of nail growth is to be attributed to a vascular factor. That the marked retardation in the denervated digits is not due solely to circulatory embarrassment is suggested by the fact that the non-denervated digits rapidly recovered their original rate of growth when the circulation was restored whereas there was no such acceleration of growth in the denervated digits. Furthermore in Case L.H. it was observed that, 18 months after the injury when nerve function had been fully restored, the nail growth on the two sides was the same. At this time, however, there were signs of a persisting circulatory defect.

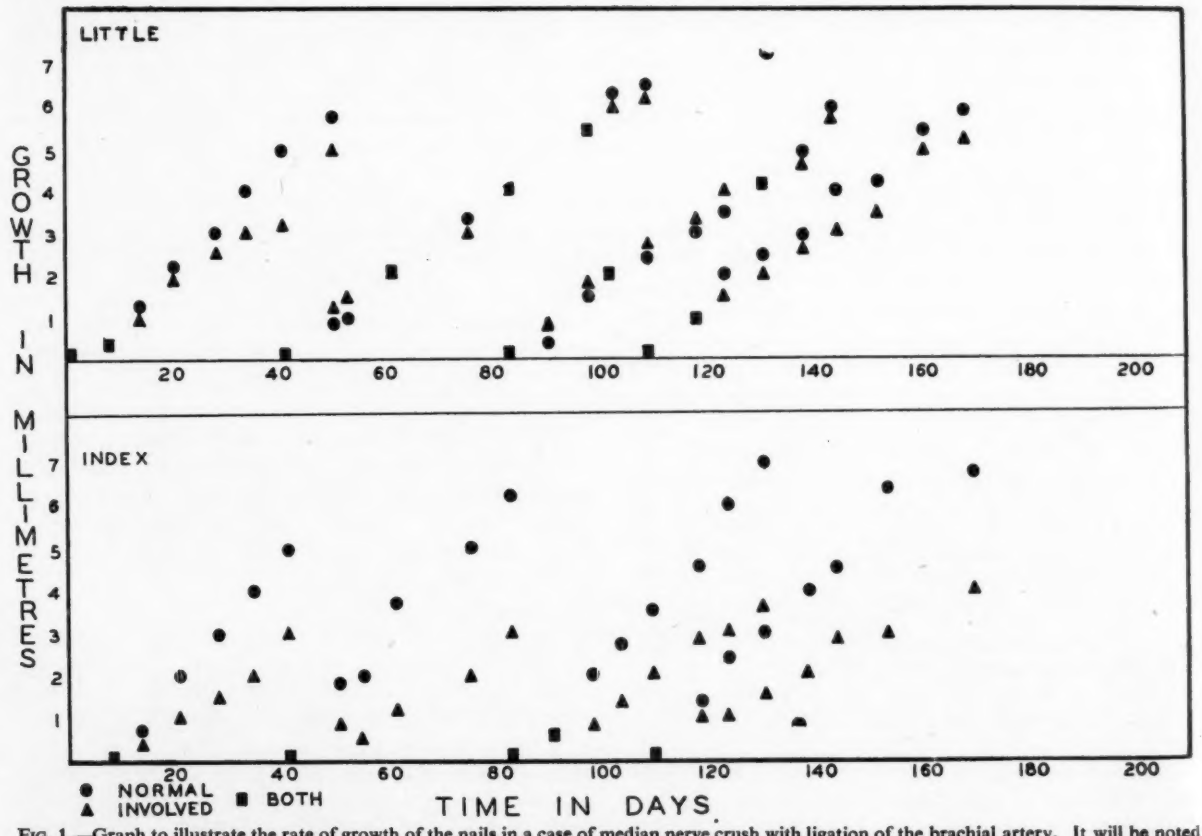

FIG. 1.—Graph to illustrate the rate of growth of the nails in a case of median nerve crush with ligation of the brachial artery. It will be noted that there is a marked difference in the rate of growth on the two sides for the index finger and virtually no difference for the little finger.

There appeared to be a further slowing of nail growth during the winter months in two cases. Owing, however, to the limited number of patients available for observation, the data were insufficient for a precise assessment of the influence of the seasons on nail growth in denervated digits.

Summary

After uncomplicated nerve injuries nail growth in the denervated digits was retarded in some cases but not in others. When growth was affected the retardation was only slight.

When arterial ligation was superimposed on the nerve injury growth was consistently and markedly retarded in the denervated digits, greatly exceeding the slight slowing recorded after uncomplicated nerve injuries. There are three reasons why this greater slowing is not to be attributed solely to the vascular injury: (*a*) it outlasted the period of circulatory embarrassment; (*b*) the non-denervated digits of the involved hand were not affected; (*c*) complete nerve recovery resulted in an equality of growth in the involved and corresponding normal digits.

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J. Neurol. Neurosurg. Psychiat., 1952, **15**, 54.

MEMORY DEFECTS AFTER HEAD INJURY

BY

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The phenomenon of retrograde amnesia following head injury has attracted considerable interest in recent years. Retrograde amnesia, usually of short duration, is almost invariably present in cases of closed head injury of sufficient intensity to produce some period of post-traumatic amnesia. Although the duration of retrograde amnesia seldom exceeds a few minutes, it may in more severe cases embrace a period of hours or even days. In general, the duration of permanent retrograde amnesia tends to be greater in the more severe cases and to be roughly proportional to the length of post-traumatic amnesia (Russell, 1948b). There are, however, exceptions to this rule (Symonds, 1949). Other features of retrograde amnesia which have been studied in recent years include its manifest " shrinkage " during recovery from the post-traumatic confusional state (Symonds, 1949 ; Paterson and Zangwill, 1944) ; the occasional presence of " visions " or islands of memory within the period of amnesia (Russell, 1932, 1948a) ; and its reduction, in some cases at least, by investigation under barbiturate hypnosis (Russell and Nathan, 1946). These various features of traumatic amnesia have been well described by Russell and Nathan (1946) and do not call for further comment.

In addition to the residual retrograde amnesia, even where this is extremely brief, there may be a further weakness in the memory for events which have occurred before the injury. Although such defects have often been recognized, little attention appears to have been given to their systematic study. Our intention in the present paper is to analyse the incidence and character of these memory defects in a group of patients who appeared to have made a complete recovery from head injuries, and to indicate that in some cases, despite lack of residual impairment on tests of memorizing and intelligence, faulty recall of pre-traumatic events may be serious enough to constitute a minor residual disability.

Cases

The nature of memory impairment for events shortly preceding concussional head injury is well illustrated in the following cases.

Case 1.—J. H., aged 17, a laboratory assistant (R.I. No. 104025), fell from his motor-cycle on November 24, 1950, sustaining a mild concussional head injury. On admission to hospital an hour later he appeared fully conscious and orientated in all spheres. Recovery was uneventful, and the patient was discharged 19 days later free from symptoms. Retrograde amnesia was of a few seconds and post-traumatic amnesia a matter of minutes. There was no evidence of post-traumatic intellectual loss nor any defect on tests of memorizing.

The patient was interviewed by one of us (M. W.) on several occasions during his stay in hospital and after his return home. It was found that he was able at all times to give a clear account of the greater part of the course of his journey before the accident. His last memory was of his machine skidding, though he did not recollect his actual fall. At the same time, it was found that there were amnesic gaps in his memory for parts of the journey before this episode. Thus, he could not remember driving along a stretch of some five miles of road to the spot where the accident occurred, and no amount of effort enabled him to recover this memory. If retrograde amnesia is assessed as " seconds ", therefore, it is important to bear in mind that before his last memory there is an amnesic gap covering several minutes, and that his memory for the journey as a whole was patchy.

This case is of interest in so far as the brief permanent retrograde amnesia bounded by the patient's memory of his skid was associated with a further amnesic gap covering a longer period. In the following case, likewise a comparatively slight injury, the residual amnesic defects were both more complex and more severe.

Case 2.—R. H., a motor mechanic aged 20 (R.I. No. 132125), fell from his bicycle on October 18, 1950, hitting the left side of his head and sustaining a concussional head injury. On admission, he was conscious and orientated, though drowsy, and had a confused recollection of the preceding half-hour. Recovery was

uneventful, and the patient was discharged after 12 days with residual retrograde amnesia of "seconds" and post-traumatic amnesia of under one hour.

He was interviewed several times during his stay in hospital, and after discharge. On the morning after his accident he had recovered a vivid recollection of the moments immediately before the accident. He had a clear memory of rounding a corner into a main road, of seeing a bus drawn up just ahead, of hitting the kerb and flying over the handlebars. He could not, however, recollect where he had been going at the time, nor for what reason. Nor, on further examination, could he describe any incidents during the previous seven days. Three days later he was able to recall that some time during the week before the accident he had been moved to a different job. He also recovered some vague recollections of events which had occurred on the day of the accident itself, but apart from these isolated recollections his memory for the week before the accident up till the few moments just preceding it was very defective.

Case 2 provides a good example of a patient who, despite typically brief retrograde amnesia, presented quite marked residual memory defects for events preceding the accident by some days. This patchy amnesia had not recovered when the patient was seen three weeks after the accident.

In the following case the residual memory defects were considerably more severe and gave rise to a genuine minor disability.

Case 3.—P. H., an agricultural labourer aged 23 (R.I. No. 134424), ran into a telegraph pole on his motor-cycle when returning home from work. On admission (December 4, 1950) he was unconscious, with a skull fracture in the right parietal region. His condition deteriorated after a few hours, and at operation a right extradural haematoma was evacuated. Following a number of epileptic attacks, further extradural and subdural clots were removed at operation eight days after admission. Apart from a left hemiplegia, which improved steadily, his neurological condition was satisfactory. On discharge, four and a half months after injury, he showed no impairment on tests of memorizing, learning, abstraction or construction. Retrograde amnesia was assessed as 24 hours and post-traumatic amnesia as seven weeks.

When interviewed by one of us (M. W.) three months after discharge, the patient was able to recollect several events which had occurred the day before the accident, when he had taken part in a motor-cycle trial. He had a vivid recollection of the intense cold and of drinking rum to keep warm. At the same time, his memory for a period before this was grossly impaired. Thus, he was unable to recall that he had been off work with a mild attack of influenza during the greater part of the week before the accident, although this event was recalled and recounted by his parents. He was also unable to recall much of his most recent work. According to his parents, he had been looking after a litter of pigs for the month or so before the accident and had taken home daily descriptions of their progress. The patient was unable to recall the pigs at all, his last recollection of farm work being that of carting sugar beet two months previously.

The discrepancy in this case between the events remembered by the patient and those by his parents indicates that the patient's haziness of memory for pre-traumatic events cannot be ascribed to ordinary forgetting with lapse of time.

It will be noted in the above account that, although the patient's most recent memory dating from before the injury related to the day preceding it, there was none the less considerable amnesia for events which fell within the previous few weeks.

Similar phenomena observed in three further cases may be communicated more briefly.

Case 4.—K. G., a girl aged 22 (R.I. No. 73415), fell from her bicycle, sustaining a mild concussion. Post-traumatic amnesia was 24 hours and retrograde amnesia a few seconds. Her last memory, which was very vivid, was of a cat running across the road a few seconds before the accident. As in Case 2, however, the patient testified to complete amnesia for the circumstances and general intention of her journey. She knew where she was and could recall how she had arrived there, but was unable to say with what intention she was making the journey or where she was heading for. These were still unknown to her two weeks after discharge.

Case 5.—E. C., an ambulance driver aged 38 (R.I. No. 141508), sustained a frontal fracture as a result of a motor-cycle accident. On discharge after four weeks in hospital, post-traumatic amnesia was assessed as 12 hours and retrograde amnesia as 30 minutes or less. He could describe very precisely a visit to a friend's house only half an hour before the accident and the full circumstances surrounding it. On the other hand, memory for the week preceding the accident was very defective, and the patient insisted that this was not in keeping with his normal powers of recollection. He was unable to give any details of his activities during the Whitsun holiday three days before the accident, and qualified any statements he did make with phrases such as, "I think I went . . . ", and, "I have a strong suspicion I did". In addition, his memory for the order of sequence of several journeys which he made during the week in question was exceedingly vague, whereas his memory for similar journeys made in previous weeks was correct. In his own words, "Normally I'd be able to tell you what I had picked up, who, and where. But last Tuesday we had rather a busy night; I know it included one accident, but I can't tell you where it was."

In this case, therefore, there was a patchy amnesia covering about a week in addition to the complete retrograde amnesia of 30 minutes or less.

Case 6.—T. F., an engineer aged 40 (R.I. No. 131865), sustained a mild head injury which gave rise to an intracerebral haematoma. He was confused and amnesic for some weeks after operation, and on recovery retrograde amnesia was assessed as one week and

post-traumatic amnesia as four to five weeks. At about the time of discharge, when he showed above average performance on tests of intelligence and memorizing, it was ascertained that the patient could in fact recall certain events which fell within the scope of the retrograde amnesia (" islands " of memory). Some excursions undertaken by himself and his wife during this time, details of which were given to us by the latter, were also described adequately by the patient when he was directly asked about them. He had not referred to them before, however, because they appeared to him to date from a much earlier and ill-localized period of his previous life. Their proper time-reference was only established firmly after several conversations with his wife.

Analysis of Memory Defects

The principal dysmnesic features (other than retrograde amnesia and post-traumatic amnesia) shown in this series of cases are as follows :—

Memory Gaps.—Gaps in memory for events leading up to the accident, yet not falling within the duration of the retrograde amnesia, were shown most clearly by Cases 1 and 3. These gaps remained after retrograde amnesia had shrunk to its final brief duration and did not appear to be spontaneously recoverable.

Defective Recollection.—Hazy and ill-defined recollection of pre-traumatic events which had occurred up to a week or more before the accident was noted in Cases 2, 3, and 4. In one of these cases at least retrograde amnesia as conventionally assessed was extremely brief. The forgotten events, as far as could be judged, do not extend far back into the remote past.

Time Errors.—Disturbances in the recall of sequence of recent events was noted in Case 5. In Case 6 some disturbance of the " sense of pastness " (Gillespie, 1937) was apparent.

There seems to be little doubt that these lapses are due to genuine defects of memory and are not explained by normal forgetting. This is clear, first, from the fact that the patients themselves often later recover memories for events at first thought to have been forgotten, as in Case 2 ; and, secondly, from comparing accuracy of recollection of the same events by the patient and his relatives or friends, as in Cases 4 and 6.

It also seems clear that the defects are associated with the head injury itself and not with either the accident or subsequent stay in hospital, although amnesia arising from fright has been reported (Cairns and Holbourn, 1943). In one instance a husband and wife, both highly intelligent people, were involved together in a car accident. The wife sustained a mild head injury, with retrograde amnesia and post-traumatic amnesia of some hours, and a diffuse patchy amnesia for events preceding her " last memory " by some days. Information about her activity was readily supplied by the husband, who, though seriously injured, sustained no concussion and had no difficulty in recollecting these events.

Incidence of Memory Defects

A further study has been made of 24 cases of closed head injury of varying grades of severity, from the point of view of residual memory defects of the kind described. Only patients from whom adequate information could be obtained and verified and who proceeded to a full intellectual recovery have been included in this series, which thus constitutes a small group selected from the many initially observed. The cases have been divided into three groups on the basis of duration of post-traumatic amnesia, and hence of presumptive severity of injury (Symonds and Russell, 1943). The incidence of memory defects falling into any of the categories listed above and their distribution in the three groups of subjects is shown in Table I.

It will be seen from Table I that two-thirds of the cases gave evidence of a memory defect extending beyond the duration of retrograde amnesia as clinically ascertained. There is some suggestion that the incidence of such defects is more frequent in the more severe injuries (Groups 2 and 3).

TABLE I

DISTRIBUTION OF MEMORY DEFECTS

Group	Cases	P.T.A.	Residual Memory Defects	
			Present	Absent
1	8	> 1 hr.	3	5
2	8	1–24 hrs.	7	1
3	8	1 week or over	6	2
	24		16	8

P.T.A. = post-traumatic amnesia

The relationship of residual memory defect to duration of retrograde amnesia in this series of cases was likewise studied and is shown in Table II. The figures suggest that, regardless of the length of post-traumatic amnesia, patients with retrograde amnesias longer than a few seconds are more likely to have other memory defects of the type described than are those with retrograde amnesia lasting only a few seconds.

TABLE II

RELATIONSHIP OF RESIDUAL MEMORY DEFECT TO DURATION OF RETROGRADE AMNESIA (R.A.)

Duration of R.A.	Group 1		Group 2		Group 3	
	Memory Disturbance	No Memory Disturbance	Memory Disturbance	No Memory Disturbance	Memory Disturbance	No Memory Disturbance
Seconds ..	5	3	2	1	1	2
Up to 1 hr. ..	—	—	4	0	1	0
Over 1 hr. ..	—	—	1	0	4	0

Discussion

Retrograde amnesia of long duration has commonly been observed in the post-traumatic confusional state, and its gradual " shrinkage " during recovery is a common-place. Although this " shrinkage " is most obvious during the later stages of confusion (the so-called Korsakoff phase), it not uncommonly continues after the period of post-traumatic amnesia is at an end (Symonds, 1949). Our observations make it clear that even after the retrograde amnesia has shrunk to its final (usually brief) duration, memory for events preceding the injury is not necessarily intact. When the residual retrograde amnesia is extremely short, as in Cases 1, 2, and 4, closer enquiry may disclose further amnesic gaps or haziness in recollection of events preceding the injury by days or even weeks. In some cases, e.g., Cases 5 and 6, events may be recalled but their localization in time is defective. This is, of course, a common feature in the milder amnesic states. In view of these considerations, it may be concluded that the full extent of memory defect for events preceding the injury is not necessarily given by the duration of retrograde amnesia as conventionally ascertained. This does not mean, however, that the usual means of assessing retrograde amnesia are necessarily unreliable. It appears more likely that there are two separate aspects of the memory disturbances following head injury : (1) the short and usually complete retrograde amnesia ; and (2) more diffuse and widely distributed disturbances of memory for pre-traumatic events.

The latter have their parallel in observations which have been made of residual amnesias following electro-convulsive therapy (E.C.T.). Although the duration of post-convulsive retrograde amnesia is as a rule well under a minute (Mayer-Gross, 1943), more extensive memory defects are not uncommonly found. Cohen (1939) has called attention to retrograde amnesia covering a period of up to a day or more, but recovering spontaneously within a few hours. Delay (1943) has noted, in addition to retrograde amnesia of variable duration, isolated disturbances in general memory for information and events which may persist for a considerable period. Similar observations have been reported by Brody (1944) in a small number of cases. More recently, Janis (1950) has demonstrated by means of special techniques, extensive disturbances in the recall of well-established personal information after E.C.T.

Although the amnesias reported after E.C.T. were somewhat resistant, they were regarded, in principle at least, as reversible. In this paper we have only described retrograde memory disturbances which showed no improvement after several weeks. In addition, however, we have observed a large number of patients in whom, although the actual duration of the retrograde amnesia has not shortened, there has been considerable improvement in memory for events on the threshold of the amnesic gap. Although in the instances observed here the memory defects concerned events of fairly recent origin, it is possible that with more extensive means of enquiry and verification disturbances of very remote memory might also be discovered. These amnesias, however, do not seem to be due so much to a genuine abolition of the memory traces as to a blocking of recall which can be removed by certain circumstances such as conversations with relatives or return to the pre-traumatic environment. One of our patients told us that in the weeks following his discharge a meeting with a friend or a visit to a place known previously would often serve to bring back memories of recent experiences which he had been unable to recollect since his injury. In view of these considerations, it may be suggested that residual memory defects of the type described in this paper are severe grades of reversible disturbances and due to impairment of recall rather than absolute amnesia. In this respect they appear to differ from the absolute retrograde amnesias lasting a matter of a few seconds, which may be due to

failure of registration in the first instance and are probably not recoverable. Absolute retrograde amnesias of longer duration may be due to a combination of both inaccessibility and lack of registration. In certain cases they have been shown to behave like the more diffuse disturbances of pre-traumatic memory, in that certain aspects are recoverable. The work of Russell and Nathan (1946) suggests that some yield to investigation under barbiturate hypnosis, while Klein and Kral (1933) have reported a traumatic retrograde amnesia reduced by reconstructing the situation which had obtained shortly before the accident.

It is apparent from these observations that, besides the shrinkage of absolute retrograde amnesia seen during recovery, which has been found by Russell and Nathan (1946) to follow a time sequence, there is a " filling-in " of more extensive retrograde memory disturbances which can continue after the absolute retrograde amnesia has reached its static level, and in which other factors as well as time are of importance.

The anatomical lesion associated with the incidence of retrograde memory disturbances is obviously a question of great importance. Little can be said on this score from the small number of cases reported in this paper, but it is clearly a subject which merits further investigation.

In conclusion, it may be noted that the presence (or absence) of residual memory defects for pre-traumatic events might have some value as an additional criterion in assessing the progress of recovery. When taken in conjunction with the respective duration of retrograde and post-traumatic amnesia, it might lead to a more accurate appraisal of the extent of general cerebral disturbance. It is also possible to regard such memory defects as constituting the mildest grade of post-traumatic intellectual loss. Should they appear in more outspoken form, it may be surmised that recently acquired information, knowledge, and skills would be appreciably affected.

We are greatly indebted to Sir Hugh Cairns for his help and encouragement in this study ; to Dr. Ritchie Russell and Mr. W. Lewin for valuable advice and criticism ; and to the Accident Service of the Radcliffe Infirmary, under the direction of Mr. J. C. Scott, for permission to study the patients under their care.

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J. Neurol. Neurosurg. Psychiat., 1952, **15**, 59.

PULSE-WAVE ELECTROCOMA

BY

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There have been many articles dealing with the clinical value of electrocoma, as we prefer to call "electronarcosis", in the course of the last half dozen years. Some writers make considerable claims for the technique in the treatment of schizophrenia and other mental disorders, but a few have been less favourably impressed. This contribution to the literature is intended to be technical and methodological rather than clinical.

Although we have been treating patients by electrocoma for the last four years, we do not feel entitled yet to express a firm opinion as to the value of the treatment because (1) the total number of patients treated at St. Bartholomew's Hospital to date, although "respectable", is insufficient for statistical purposes; (2) all our patients have been treated under out-patient conditions, and none have received more than one treatment a week; (3) there is no means of judging whether a series of electrically induced convulsions given at the rate of one a week would have produced the same clinical results as electrocoma. The only method of arriving at an opinion on this point would have been to have treated a number of patients by electroplexy and another clinically comparable control group by electrocoma and to have assessed the results accordingly. We were not in a position to do this. We must be content, therefore, to record a clinical impression to the effect that certain patients, notably early cases of paranoid schizophrenia and the obsessional form of psychosis, have derived benefit from the treatment. We have noted improvement in a number of patients exhibiting phobic anxiety states with obsessional features. And the results are sufficiently encouraging to warrant continued investigation.

It is some years now since Frostig, Van Harreveld, Reznick, Tyler, and Wiersma (1944) applied direct current, interrupted direct current, and alternating current to the brain, and later, together with Tietz, Thompson, Van Harreveld, and Wiersma (1945) evolved the technique which came to be known as electronarcosis.

Their first experiments were conducted with dogs and made use of an interrupted direct current produced by a thyratron-excited square-wave generator. They found that they could induce coma with a pulse of about 140 microseconds' duration with a repetition-frequency of anything from 120 to 1,000 per second. If alternating current is used, frequencies of from 30 to 8,000 cycles per second are effective, but the coma is more disturbed with frequencies that exceed 500.

Some six years ago Tietz made use of an apparatus which passed an alternating current of mains frequency through the brain in such a way that the current level could be pre-set and maintained in spite of the rapid changes in the patient's impedance. Tietz's original circuit included a power amplifier with an appropriate negative feed-back, but in our original version of this apparatus we used a high-voltage source of alternating current and a large series ballast resistor. Our object was to prevent changes in the patient's resistance materially affecting the total impedance of the circuit, thus enabling the current to be maintained at the desired level. Others have used magnetic stabilizers to attain the same end (MacPhail, 1949).

Although all these apparatus deliver 50–60 cycle alternating current to the patient and can produce and maintain coma for minutes at least, the risks involved impressed us as being unnecessarily high: respiration is apt to be seriously embarrassed, with consequent stridor and cyanosis; there is a dramatic rise of blood-pressure, often as much as 30 or 40 points; cardiac irregularities, including fibrillation, are temporarily produced; a condition amounting to tetany requires to be maintained if a satisfactory coma is to be kept up; it is almost impossible to produce or maintain coma without a preliminary convulsion. This is not a disadvantage in itself, but it makes it extremely difficult to decide whether the clinical results of the treatment are to be attributed to coma or to electroplexy (E.C.T.).

We had one death from what appeared to be central heart failure during coma induced by a

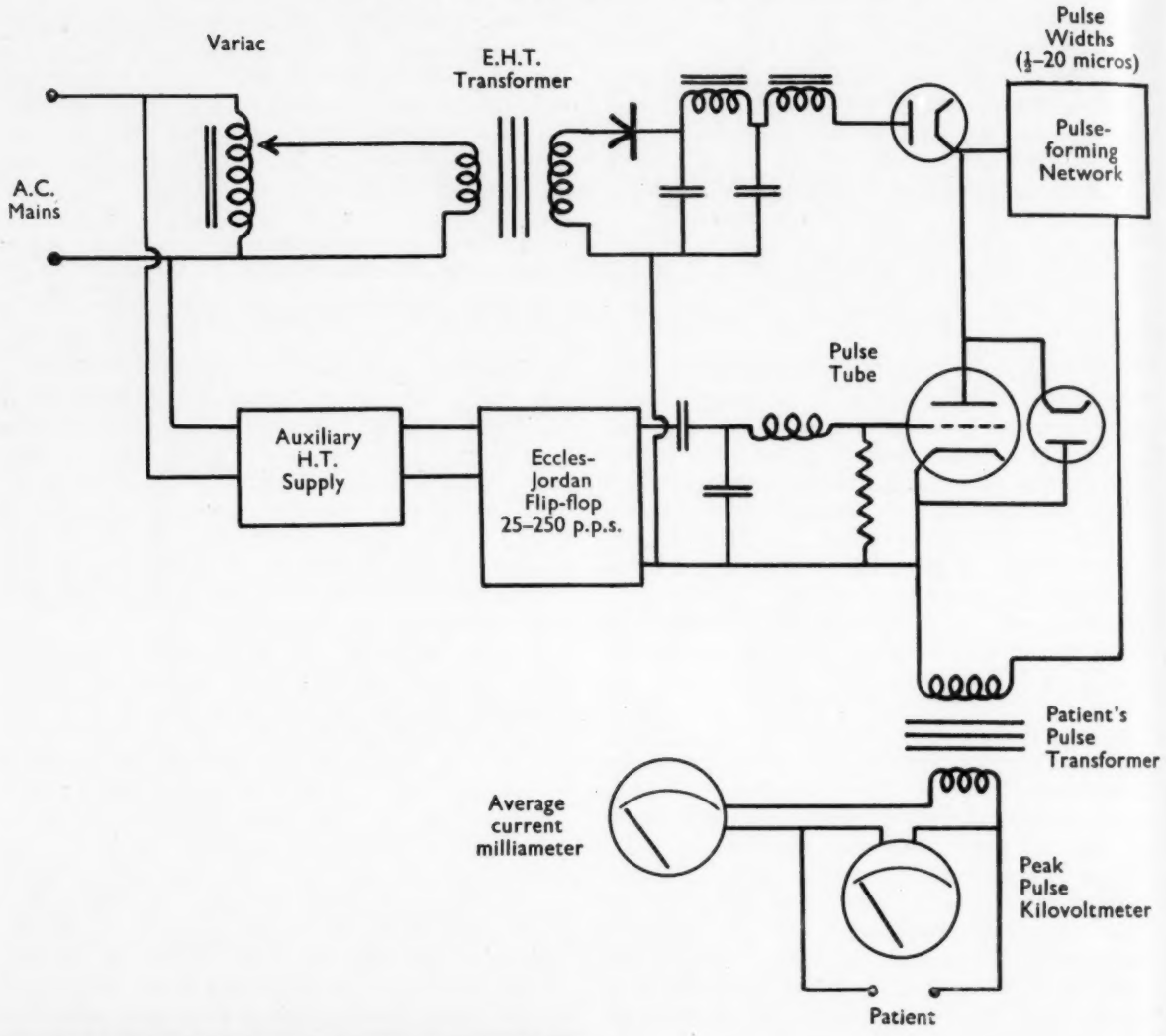

FIG. 1

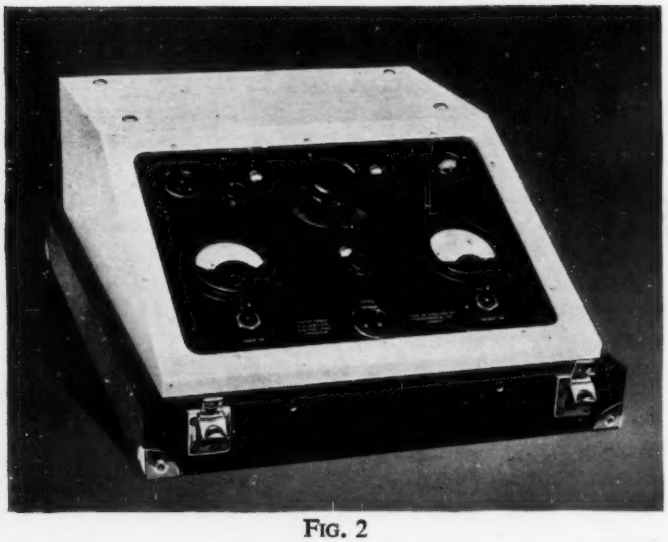

FIG. 2

50-cycle current. The patient, a young leucotomized woman, had had three previous inductions of electrocoma and appeared to be benefiting from the treatment. All efforts at resuscitation by the anaesthetists in attendance proved unavailing. We therefore decided to experiment with a wide range of electrical wave-forms in an attempt to induce coma with a large margin of safety for the patient and as little anxiety as possible to the operator: we wished to reduce the physiological side-effects to a minimum compared with those which seemed inseparable from the use of raw alternating current.

We finally decided on trains of pulses of from ½–20 microseconds with repetition-frequencies of 50–200 per second as being the most desirable.

The peak pulse voltages applied to the patient range up to 3 kilovolts. This range represents previously unheard-of peak voltages and currents, and induces an entirely new set of electrical reactions and demonstrates that the human brain can with complete safety support high-voltage currents of very short pulse duration continuously. The watt-second rate is the determining factor.

The electrical circuit used in this work is illustrated in Fig. 1. It consists of a high-voltage transformer fed from the A.C. mains through a " variac ". A direct high tension supply is made available by rectifying the output of this transformer, and smoothing by means of condensers and choke.

An auxiliary high tension supply is fed to a modified Eccles-Jordan flip-flop oscillator. This drives the main pulse-tube which generates high voltage pulses to the pulse-forming network, finally delivering to the patient who acts as the load. Pulse voltage is variable from 0 to 2·2 kilovolts ; the pulse width from 0·5 to 20 microseconds. The peak pulse kilovoltage is shown on an electrostatic voltmeter. The average current to the patient during treatment is indicated by a moving coil milliammeter. The energy factor is selected by a rotary switch which also regulates the pulse width in microseconds. The outward appearance of the apparatus with its carrying cover removed, showing the control panel, can be seen in Fig. 2. This instrument, the " pulsacon ", is manufactured by Messrs. Theratronics, Ltd., 6, Argyle Walk, London, W.C.1.

Technique

Preparation of Patient.—The patient should have had no food for the previous four hours. He is given 1½ gr. " epanutin " by mouth about an hour before treatment and a subcutaneous injection of atropine sulphate, gr. 1/100, about half an hour later. The same sort of couch and hard pillow for hyperextending the back as are used in electroplexy are employed in electrocoma, and, as in electroplexy, a solid rubber gag is placed between the patient's teeth.

A tight-fitting rubber bathing cap (we have found this to be the most suitable form of head-gear) is fitted over the head in such a way that the anterior edge reaches the line of the eyebrows. The patient is now lightly anaesthetized with an intravenous barbiturate. We find that 0·5 g. thiopentone usually suffices. The electrodes are now placed in position under the bathing cap. Each consists of a foil of stainless steel, 1⅝ in. by 1⅜ in., sandwiched in ¼ in.-thick slabs of cellulose sponge. The sponge and the metal electrode are retained in position by being sewn into a little envelope of lint. Each electrode unit is soaked in lukewarm 10 to 20% saline. The correct position of the electrodes is such that the lower lengthwise edge is ½ in. above the mid-eyebrow level, with the proximal anterior edge of the electrode half-way between the mid-pupillary line and the outer canthus of the eye (Fig. 3).

FIG. 3a

It is important that the electrodes should be accurately placed, as too low a position is likely to cause excessive laryngeal spasm, and a too medial position produces too light a coma. Counter-pressure is applied to the shoulders and hips, although the muscular contractions resulting from

FIG. 3b

the initial stimulation are less tetanic and still less tonic than in electroplexy. In this connexion, it is worth noting that out of a total of some 500 treatments only one (doubtful) lamellar crush was observed, and this occurred in the case of a very muscular young man who had not been anaesthetized with thiopentone.

Electrical Dosage Values.—We usually induce coma with the following electrical values : 1·5 Pkv ; pulse repetition-frequency, 105 per second ; pulse-width, measured in microseconds, 10 ; milliamperage, 9. The current is passed for 30 seconds, the patient having been previously oxygenated by the anaesthetist. There is an immediate contraction of skeletal muscle which is sub-maximal and tetanic in character, but cyanosis is not observed. The contracted muscles make it impossible to feel the pulse at the wrist or at the neck, but the femoral pulse is not affected. There is an immediate rapid rise in the pulse rate. The apex beat is frequently obscured, and the heart sounds are often inaudible on auscultation.

The current is reduced after the first half-minute by diminishing the pulse-width and lowering the kilovoltage, which rises automatically as the pulse width is reduced, so as to keep the kilovoltage for a while at its original level of 1·5 Pkv. This may be reduced later to 1·3 Pkv. The pulse-width is reduced from its original level of 10 microseconds to 3·3 microseconds, or sometimes, especially in the case of muscular young men, to 5 microseconds. The milliamperage falls proportionately to the current and the kilovoltage and will now be about between 3 and 5 m.a. Respiration is spontaneously re-established within 10 to 20 seconds, frequently with some stertor or stridor owing to laryngeal spasm. The respiratory rate may be as high as 40 per minute at this stage although it is more commonly under 35. The pulse rate may be as high as 160 for a short time, but usually falls rapidly. Dropped beats and extra-systoles often occur at this stage of treatment.

Diminution of pulse-width is followed by a reduction of muscle tone. With a pulse-width of 3·3 microsecs. an observer can with ease effect passive movements of the limbs. Further reduction of the pulse-width, say to 1 microsec., is accompanied by considerable generalized hypotonia and relaxation.

The pulse rate is usually high throughout coma ; this would appear to be a central tachycardia, as, after the first 30 seconds of induction, there is no cardiac or respiratory strain or embarrassment. When the current is finally switched off, the pulse rapidly returns to normal.

From this stage of the treatment onwards the pulse-width is slowly reduced to the lowest value consistent with the continuation of coma, maintaining the voltage at the 1·5 Pkv. level. It will be remembered that the milliamperage varies automatically with the pulse-width.

When the electrically induced coma has been maintained for from one to four minutes, the patient may become slightly restless and begin to phonate. This does not appear to be identical with the hyperkinesis described by Frostig ; it is more likely to be a manifestation of subcortical activity. Further reduction of the electrical stimulus in terms of kilovoltage, or preferably pulse width, restores tranquillity and allows coma to proceed smoothly. Oxygen, which is given before and during induction, can usually be discontinued after the first 45 seconds.

The time during which smooth coma may be continued varies from patient to patient. Powerfully built young males as a rule require a greater electrical dosage than others, and even then they frequently come out of coma after four or five minutes, and rarely remain comatose for longer than eight minutes, whatever the stimulus. Slightly built, young female patients are the easiest to keep comatose and the coma may be maintained for up to a quarter of an hour, if desired. It is generally agreed that the optimum length of treatment is seven minutes.

When coma is prolonged neurological signs similar to those associated with decortication may be observed ; for example, the posture is decorticate, the plantar responses are extensor, and the knee- and ankle jerks tend to be increased. These signs disappear quickly and are no longer to be elicited two minutes after the cessation of coma.

The ordinary criteria by which one judges the depth of anaesthesia do not apply in electrocoma ; the respiratory rhythm is different from that associated with anaesthesia produced by other methods, expiration following inspiration without pause, the whole respiratory sequence being firm and forceful. The eyelids remain tightly closed, and lachrymation may occur as the result of local radiation of the current to the muscles of the eyelids. If the eyelids are prised open within 30 seconds of the thiopentone injection or at any time during coma, the pupils are seen to be contracted. Termination of coma is soon followed by dilatation of the pupils.

At first, we interpreted the type of phonation as evidence of lightness of anaesthesia or impending termination of coma. We no longer believe this to be the case, as even a slight reduction of the electrical stimulus abolishes this sort of subcortical

activity. If the treatment is artificially interrupted at this stage, the patient does not immediately regain full consciousness but falls asleep for up to 20 minutes or more.

The likelihood that this pseudo-purposeful behaviour of the restless kind is subcortical rather than cortical in nature is supported by observations on patients who were given electrocoma without thiopentone anaesthetization. Some of these patients, who were given an electrical stimulus which was insufficient to produce smooth coma and exhibited restless motor patterns, subsequently described subjective experiences of a crude, emotionally over-invested nature which were too bizarre to be properly verbalized. One patient, for example, described great balls of fire and a feeling as though he were approaching the Judgment Seat ; another patient was aware of increasing tension amounting to horror. A third patient became so conditioned that he would fly from the sight of electrical apparatus no matter where it was. On one occasion he saw some electrical appliances being unloaded from a military lorry and was quite unable to restrain himself from making a dash into the road. These conditioned reactions do not occur with thiopentone.

Summary and Conclusions

Although we are not yet prepared to assess the clinical value of electrocoma in psychiatric practice, it is, in our opinion, a form of electrocerebral treatment which should continue to be investigated.

We believe that a pulse-wave current allowing of four variables—pulse-width, repetition frequency, peak voltage and milliamperage—provide the best and safest method of inducing and maintaining coma.

An apparatus which fulfils these requirements and its method of employment are described in detail.

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J. Neurol. Neurosurg. Psychiat., 1952, **15**, 64.

PROLONGED COMPRESSION OF THE BRAIN RESULTING FROM AN EXTRADURAL HAEMORRHAGE

BY

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With the exception of the present case, our experience over the past 10 years in cases of compression of the brain by extradural haemorrhage has been that, once the compression has been initiated, it is speedily progressive, and ends fatally within 48 hours if not surgically relieved before irreversible changes have developed in the brain stem. The object of the present communication is to put on record the unusual sequence of events that occurred in one case of middle meningeal haemorrhage.

Case History

A coal miner, aged 47 (Case No. 14698), at 8 a.m. on November 11, 1950, was thrown from his pedal cycle on to his head following a collision with a motor car. Unaided, he picked himself up, and, though slightly dazed, was able to make his own way home. He remained fully conscious till mid-day when he became drowsy and began to vomit. He was admitted to the Neurosurgical Unit at 5 p.m. the same evening.

On admission, the man was found to be in a state of deep confusion bordering on semi-coma. On loud and repeated questioning, he would respond, and point to the back of his head and moan with pain. Undisturbed, he immediately went off to sleep. Apart from his mental state, no other abnormal neurological signs were elicited, though it is possible that he might have shown a defect in the fields of vision had it been possible to chart them. There was a large bruise at the back of the head on the left side. There were no injuries elsewhere on the body. Radiographs did not reveal a fracture of the skull. The pulse rate was 50, the blood pressure 150/80 mm. Hg, and respiration was normal in rate and rhythm. Though a surface haemorrhage was diagnosed, it was decided not to operate immediately, chiefly because there were no definite localizing or lateralizing signs, apart from the bruise on the head.

By the following morning there had been no change in the patient's depth of unconsciousness. By November 16, his mental state had improved to that of slight confusion : he sat up in bed and fed himself, but he complained of persistent generalized headache. Improvement was maintained for about another week when his headache intensified, and he became a little more confused.

On November 27, papilloedema was observed for the first time. Air encephalography showed the ventricular system to be pushed over to the right with flattening of the body of the left ventricle. Since the encephalogram did not adversely affect his mental state, we decided to let him get over the effects of this manoeuvre and did not in fact elect to operate on him until December 8, that is, about one month after the injury.

Operation (G.F.R.).—Under general anaesthesia a large osteoplastic flap was turned, centred just behind the external auditory meatus. On raising the flap a large blood clot was encountered in the extradural space. It was at least 4 in. long × 3 in. high × 1½ in. thick in its central part. Covering the surface of the clot was a thick membrane such as one sees in chronic subdural haematomata, while the contents consisted largely of dark, watery fluid, such as is found in chronic subdural haematomata, and quite different from the mixture of bright red blood and gelatinous blood clot seen in acute middle meningeal haemorrhage. Though the blood clot was easily removed, bleeding was difficult to control, because of persistent oozing from multiple vessels in the capsule of the clot. Indeed, so persistent was this bleeding that the wound had to be lightly packed with gauze to control it. The wound was then closed in the usual way with an end of the gauze issuing from one corner of the wound. The gauze pack was removed 48 hours later.

On the morning after the operation the patient's condition was obviously improved, and his headache had gone, never in fact so far to return. By the second day his mental state was normal, and he had no complaints.

He was discharged from hospital on December 20, 1950, without complaint or disability apart from a small operative defect on the left side of the skull. He returned to work in February, 1951, as an underground miner, and has worked regularly ever since. On repeat examination he has made no complaint of headache or of dizziness, and states that he feels as well and strong as ever he did.

Comment

No doubt the signs and symptoms in the first few days after the injury were due to bleeding from a posterior branch of the middle meningeal vessel. This bleeding stopped by natural processes, and as

the clot began to absorb, the patient's mental condition improved. Later on, an adventitious membrane began to form round the clot, and possibly, as the blood began to break down, dialysis occurred and an enlarged blood cyst formed with further compression of the brain causing deterioration of the mental symptoms and intensity of the headaches. Pieces of the membrane surrounding the blood clot were sectioned microscopically, but no definite structure was determinable.

Summary

To summarize, both the pathological and clinical developments in this case of middle meningeal haemorrhage were similar to those of early chronic subdural haemorrhage.

J. Neurol. Neurosurg. Psychiat., 1952, **15**, 66.

ABSCESS FORMATION IN A PITUITARY ADENOMA

BY

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The case described here is of particular interest for three reasons. First, suppuration within the sella turcica itself is an uncommon phenomenon, and suppuration within the substance of a pituitary adenoma must be extremely rare ; I can find no reference to pyogenic abscess formation in a pituitary adenoma in an extensive search of the literature over the past 20 years. Secondly, in this case no complaint of deterioration of vision had been made until the onset of what appeared to be a fulminating type of meningitis, although the adenoma must have been present for a considerable time before the appearance of the acute illness. Blindness developed with great rapidity and was complete. Thirdly, the disease process resulted in a fulminating and rapidly fatal infection with little cellular reaction in the cerebrospinal fluid.

Case History

T.R.M., a man aged 50 years, had been in excellent health until two days before his admission to the Neurosurgical Unit, Newcastle-upon-Tyne. He had been at work until October 4, 1950, when he complained of headache and began to vomit, and his temperature rose rapidly to 103°F. This was followed the next day by the rapid development of complete blindness in both eyes. At this time the clinical picture was that of meningitis : the neck was extremely stiff and Kernig's sign was strongly positive. In addition, both pupils were fixed and dilated. There was no papilloedema, but he had a left external rectus palsy. There was oedema of the left eyelid and a purulent post-nasal discharge. There is no record of the state of the deep tendon reflexes, but the superficial abdominal reflexes were depressed, and both plantar responses were extensor. Shortly after losing his vision he became unconscious, and at this point he was transferred to the Neurosurgical Unit on October 6, 1950.

On admission, the patient was semicomatose and clearly suffering from meningitis. The pulse rate was 80 per minute, and the temperature 105°F. Both pupils were widely dilated and irresponsive to light stimulation directly and consensually. The left upper eyelid was swollen and oedematous, and appeared to be ptosed. It was impossible to test his ocular movements owing to his unconscious state. There was no papilloedema and no evidence of optic atrophy. All the deep tendon reflexes were depressed ; the superficial abdominal reflexes were absent and the plantar responses were extensor in nature. Lumbar puncture showed a pressure of 120 mm. cerebrospinal fluid ; the cerebrospinal fluid was only slightly hazy and contained 154 cells per c.mm., 98% of the cells being polymorphonuclear leucocytes. The protein content of the fluid was 200 mg. per 100 ml. and there was a marked increase in globulin content. Shortly after admission the patient began to have twitchings of all four limbs which progressed into major epileptic attacks. He became deeply comatose, and died within 12 hours of admission. Radiographs of the skull were not made.

The clinical diagnosis was fulminating meningitis following cavernous sinus thrombosis presumably secondary to nasal sinus infection.

Necropsy.—A complete post-mortem examination was made by Dr. Paterson Smith, but only the findings within the cranium will be given since all other organs were normal.

There was a walnut-sized tumour occupying and protruding upwards from the sella turcica which was widely distended and its posterior wall thinned. The tumour measured 1·5 cm. antero-posteriorly, 2 cm. transversely, and protruded 1·5 cm. above the sella turcica. It expanded at its base within the sella to 2 cm. antero-posteriorly and 3 cm. transversely. The vertical measurement, or whole depth of the tumour, was 3 cm. of which 1·5 cm. protruded outside the sella. It appeared to be well circumscribed.

The infundibulum of the pituitary gland appeared to be normal. The optic nerves were flattened and stretched over its surface which showed a purulent exudate. On the transverse (coronal) section the whole left side of the tumour was seen to contain a confluent abscess from which a swab was taken for culture (Fig. 1). The main mass of the tumour had a reddish homogeneous character resembling a true pituitary tumour rather than a craniopharyngioma. No small calcified areas could be seen and there were no cysts.

Portions of the abscess area and tumour were taken for section, and the specimen was photographed.

The tumour had not infiltrated bone or brain although it had formed a cavity at the base in the region of the

FIG. 1.—The adenoma has been removed and sectioned in the coronal plane to show the extension of the tumour upwards and slightly to the right, together with the confluent abscess on the left side.

optic chiasma. Otherwise the brain was normal. There was no meningitis, hydrocephalus, or pressure cone.

Except for some mucopurulent material in the left sphenoidal air sinus, there was nothing else abnormal to be found in the skull nor was there any evidence of cavernous sinus thrombosis.

The pathological diagnosis was suprasellar and intrasellar chromophobe adenoma of the pituitary gland with abscess formation from extension of infection from the left sphenoidal air sinus which contained pus.

Histological Examination.—The tumour consisted of non-granular chromophobe cells, with a characteristic alveolar grouping and fibrous septa between the groups, though some of the cells were also arranged differently. There were also a few eosinophil and basophil cells, as well as areas of simple abscess formation containing many pus cells (Fig. 2).

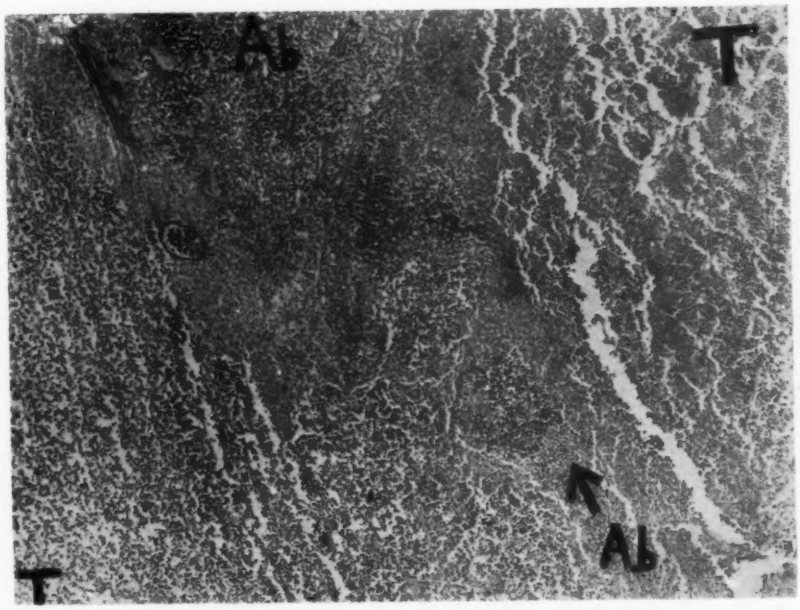

FIG. 2.—Chromophobe adenoma (T). Traversed by broad band of necrosis and inflammatory cells. (× 34).

The appearances were those of a simple chromophobe adenoma of the pituitary, showing abscess formation, apparently an extension from the left sphenoidal air sinus, which contained pus. There was no evidence of malignancy or tuberculosis. The culture obtained from the pus at necropsy showed a growth of *Bact. coli*. No organisms were grown from the cerebrospinal fluid.

Comment

This patient had undoubtedly been harbouring a pituitary adenoma for some time although he had not complained of any interference with his vision. It is of course always possible that he had a small visual field defect which had passed unnoticed, but there was certainly no evidence of optic atrophy on ophthalmoscopy. In addition there were no clinical signs of pituitary dysfunction in the rest of the body. The formation of an abscess within the tumour no doubt caused the adenoma to grow rapidly due to the mass of suppuration and also to reactionary oedema around it, thus causing rapid compression and stretching of both optic nerves. The rapidity of his death and the signs of a fulminating infection can be accounted for by spread along the infundibulum to the third ventricle together with pressure on the hypothalamus.

This patient came under my care when I was deputy regional neurosurgeon at the Newcastle-upon-Tyne General Hospital, and I should like to thank Mr. G. F. Rowbotham, the regional neurosurgeon, for his permission to publish the case record. In addition I should like to acknowledge my indebtedness to Dr. Paterson Smith, of the Newcastle-upon-Tyne General Hospital, for the pathological report.

BOOK REVIEWS

Diseases of the Nervous System. By W. Russell Brain. 1951. Oxford University Press (London: Geoffrey Cumberlege). 4th edit.; with 84 text figures. Pp. 1002. Price 42*s*.

To the general physician neurology may seem a narrow speciality: to the neurologist it appears as a broad discipline whose boundaries intermingle with many other aspects of medicine, and within which there are smaller fields of special interest to him or her. This book will serve to correct the former and to enrich the latter view—no mean achievement in a single volume which remains of manageable size and yet gives adequate reference both to classical literature and to recent work. This is the fourth edition and almost the twentieth year of publication.

The last few years have seen great activity in neurophysiology and anatomy—the scientific institutes of clinical neurology—and it is tempting to apply their findings rapidly to the interpretation of disease. Preliminary critical assessment is the more tedious but wiser course which the author has followed. The work therefore still fulfils the expressed intention of the first edition to apply anatomy and physiology to clinical problems. More use will doubtless be made in future editions of recent but now established work on both the cortex and such interesting coordinating mechanisms as the reticular formation. Photographic illustrations are good, but line diagrams are few, and there is a complete absence of illustrations of the relations of spinal cord plexuses and peripheral nerves. This is quite general to English neurological texts, but is a surprising *lacuna* when one considers how essential such anatomical knowledge is for appreciating the endless vagaries of traumatic and occupational neuritis, and how misleading some anatomical texts can be. However, these are minor points. And the combination of clinical experience and reference to the wider implications of neurological disease will ensure for the book a continued and deserved popularity amongst students, general physicians, and neurologists.

Mind, Perception and Science. By W. Russell Brain. 1951. Oxford: Blackwell Scientific Publications. Pp. 90. Price 6*s*.

Dr. Russell Brain has assembled here the various talks he has given over the past few years on the relation of mind and brain. The book is a collection of disparate essays linked by a unitary theme, the philosophically relevant aspects of the neurological background of perception and thought. The argument, illustrated rather than developed in the book, may be exemplified by a quotation from the second essay:

> "... are there events in the brain which we can understand as being at the same time both physiological and mental? I believe that there are, and it is in the realm of speech and thought that we shall find them, even though ... they are little more than dim outlines seen through the mist of our ignorance."

Dr. Brain thus appears to incline to the "double-aspect theory" of mind-brain relationship, as Professor Boring terms it in his "Physical Dimensions of Consciousness". His contribution has the merit of bringing to the discussion some of the recent findings of neurology and neurophysiology of which many professional philosophers seem unaware.

The final chapter, written specially for the book, sums up the author's general position in a "conversation" between him and the reader. This is entertainingly done, and so crystallizes the arguments in earlier essays, that it might well be read first as an introduction by the non-philosophical reader.

The book does not solve any major problems of philosophy and does not claim to. It does show that the thoughtful neurologist has a contribution to make in this field.

Diagnostic Encéphalographique. By Pierre Duran. 1951. Paris: G. Doin & Cie. With 26 text figures. Pp. 192. Price Fr. Frs. 1,100.–.

This book sets out to describe the technique of encephalography, its complications, and the interpretation of normal and pathological encephalograms. There is a chapter on the pathology of the "cephalic" cerebrospinal fluid obtained from the lumbar sac after withdrawal of fluid and air replacement. Finally there are chapters on the medico-legal and therapeutic value of encephalography.

In a work of this nature the illustrations are of paramount importance. To be easily understood, diagrams should be reduced to a minimum of lines, and those which remain must be accurate outlines of essential structures. The diagrams in this book do not fulfil these criteria; the essentials are obscured by unnecessary features, and they are not entirely accurate. Some of the photographic reproductions are poor in detail, only four "normal" films are included, and some of the common projections are omitted. The latter would be excusable if the third and fourth ventricles could be clearly seen in the normal lateral view illustrated, but this is not the case.

In the discussion of pathological conditions almost all reference to space-occupying lesions is omitted, and none are illustrated. There is no mention of the important use of encephalography in the discernment of pituitary tumours and others in that neighbourhood. There is no mention of its use in the demonstration of subdural

haematomata in sections devoted to the sequelae of trauma.

The author considers that a significant increase in the number of cells found in a specimen of cerebrospinal fluid (cephalic) obtained after the injection of air into the lumbar sac, beyond the number found in a specimen of cerebrospinal fluid taken at the beginning of the procedure, is an indication of intracranial pathology and may be a useful guide in cases of post-traumatic epilepsy, in the treatment of general paralysis, and in other conditions. No useful information was obtained from a comparison of the chemical constituents of these samples.

As a source of information this monograph is incomplete and not well balanced; it has little value as a work of reference. The bibliography contains only works by French authors, 29 out of the 76 references being to papers by one author either alone or in collaboration with others. Although mention is made in the text of papers by other workers, the references are not given. There is no adequate index.

Les Hallucinations. Clinique et Physiopathologie. By Jean Lhermitte. 1951. Paris: G. Doin & Cie. Pp. 230. Price Fr. Frs. 930.–.

This book provides an interesting account of Dr. Lhermitte's thoughts on hallucinations, a subject on which he has long been an authority. Although the book is chiefly concerned with visual hallucinations, there are chapters on other aspects which include phantom limbs, hallucinations of hearing, taste, and smell, and of movement. The various types of hallucination which may occur in organic disease of the visual pathway are well described. It is perhaps disappointing to find no serious attempt to identify those parts of the brain which must be intact for hallucinations or phantom sensations to occur, and the relationship of these areas to those essential for visual imagery and the appreciation of body image respectively. There is no index and the bibliography is incomplete and inaccurate, but in spite of these disadvantages neurologists and psychiatrists will welcome the opportunity to read this book.

L'Hypno-Analyse dans les états Schizophréniques et l'Automatisme Mental. By René Bessière and Joseph Fusswerk, with a Foreword by Professor Lhermitte. 1950. Paris: Masson & Cie. Pp. 252. Price Fr. Frs. 750.–.

This monograph brings to bear on recent methods of treatment the rich clinical tradition of French psychiatry, too little known in this country. Séglas, de Clérambault, Janet, and Ey are the outstanding contributors to the line of thought applied here to elucidate the psychopathology of schizophrenia and delusional states associated with ideas of influence. The authors have treated a number of patients, combining hypno-analysis with convulsant and other therapeutic procedures, and have attained unexpected success in many cases. Their chief interest, however, has been focused on the psychological structure revealed when their patients were under the influence of an intravenous injection of pentothal. Less persistent and less dependent on psychoanalytical interpretations than Wolberg, who carried out a similar study in the United States, they find in their patients morbid processes essentially of the same kind as those that beset normal people during adolescence, in the form of an emotional and intellectual crisis, on which Dr. Bessière has already written. The theories of psychoanalysis and of Bleuler, Minkowski, and other authorities on schizophrenia are related to these findings. It is, however, difficult to endorse the writers' opinion that hypno-analysis, which is a technique not a theory, "opens infinitely wider horizons than the Freudian doctrine": this would surely be a very difficult achievement, out-Heroding Herod.

Technique of Psychoanalytic Therapy. By Sandor Lorand. 1951. London: George Allen & Unwin Ltd. Pp. 251. Price 12*s*. 6*d*.

Dr. Lorand has much experience in guiding doctors who are being trained as psychoanalysts past the rocks and quicksands which make the last stages of "supervised analysis" hard to navigate. He gives advice on such matters as how to deal with the countertransference (the feelings that the psychoanalyst develops toward his patient), what variations upon standard procedure are permissible (e.g. allowing the patient to sit in a chair during the session instead of lying on a couch), and when to terminate treatment. Although to non-analysts the book will perhaps seem in part trivial and in part fantastic, it takes account of practical difficulties that arise frequently in this method of treatment and shows how ubiquitous sexual problems are and how determined the psychoanalyst must be to bring them into the open.

Précis de Neuro-Psychiatrie Infantile. By Dr. Gilbert-Robin, with a Foreword by Prof. Heuyer. 1950. 2nd edit., revised. Paris: G. Doin & Cie. Pp. 416. Price Fr.Frs. 1,500.–.

It is valuable to have a comprehensive account of the practice and theory of child psychiatry in France. In a country in which psychoanalysis is less entrenched than in England or the United States and the clinical tradition is strongly in favour of exact description and classification, it might be anticipated that the psychiatry of childhood would be more definite and tidy than it appears in English textbooks. This is to some extent the case: the divisions are sharper, theorizing is kept to a minimum, and the reader is spared the case histories which so often swell, without amplifying, psychiatric treatises. But neglect or ignorance of what has been going on elsewhere gives the book a parochial flavour, enhanced by the slapdash therapeutics: thus the author declares that "antispasmodics, calcium, trans-cerebral ionisation, arsenobenzols, alkaline glycerophosphates, cerebral lipoids and Vitamin B_1 have an undeniable action on the neurological substrate for speech disorders". There are, however, many illuminating chapters, which reflect peculiarly French contributions (such as those of Dupré and Wallon to anomalies of psychomotor development), and it is to be hoped that in future editions the bibliographic references will be

more carefully presented, so that original work hitherto little known outside France can be traced more readily than the book at present permits.

El Psicodiagnóstico Miokinético. Su Teoria y su Practica. By Cesar G. Coronel, with a Foreword by Emilio Mira y Lopez. 1950. Buenos Aires: "El Ateneo." Illustrated. Pp. 290. No price stated.

The study of expressive movements as a guide to personality has been advanced in Latin America by the researches of Professor Mira and his pupils. Dr. Coronel here describes fully how to administer and interpret the myokinetic test which Professor Mira first elaborated and reported during his stay in England 12 years ago. Since then it has been applied and investigated on a large scale, and its diagnostic uses explored. Apart from three introductory chapters, the author eschews theoretical questions, and his book is a manual rather than a monograph. It fulfils its purpose admirably, but there is room for a separate work collating and validating the findings of the test, at present mostly scattered through various Argentine and Brazilian journals: the literature, as the bibliography shows, is now considerable and rather inaccessible.

Die Wirkungseinheit des Lebens. By Ludwig Seitz. 1950. Berlin and München: Urban Schwarzenberg. With 54 illustrations. Pp. 500. Price D.M. 33.

The author of this philosophical work is a retired professor of gynaecology in Frankfurt, who had been led to concern himself with the problems of growth and reproduction, and so, by way of endocrinology and the nervous system, to consider fundamental issues in biology. The secondary title of the book indicates that it is a study of vital processes in health and disease, surveyed from the unitary (i.e. the cellular), the special, and finally the holistic standpoints. It is a bold undertaking, perhaps to be judged by its aim more than its achievement. Clogged and at the same time biased by his learning, which centres on the reproductive process, the author fails to attain a clear synthesis: he is also handicapped, as he realizes, by lack of contact with much recent work in the physiology and psychology of integrative processes.

Textbook of Abnormal Psychology. By Carney Landis and M. Marjorie Bolles. 1950. New York: The MacMillan Company. Pp. 634. Price 37*s.* 6*d.*

Since the first edition of this book in 1946 new material has been accumulated which made it necessary to rearrange and rewrite several sections. Its great merit lies in the avoidance of much tedious lore that appears in every psychiatric textbook, and its preference for objective studies and experiments. Although there are mistakes and peculiarities of emphasis due to the authors' lack of medical training and experience, these are compensated for by their fresh outlook and stimulating account of recent work, much of it in psychological journals that psychiatrists seldom see.

Julius Wagner-Jauregg, Lebenserinnerungen. Edited and enlarged by L. Schönbauer and M. Jantsch. 1950. Vienna: Springer-Verlag. 46 illustrations. Pp. 187. Price 17*s.* (bound 20*s.* 6*d.*).

All those who enjoy reading medical biographies will be pleased to have the one of Wagner-Jauregg (1857-1940). We associate his name with the discovery of malaria treatment for general paralysis of the insane, for which he was awarded the Nobel Prize in 1927. Apart from the account of personal experiences which this biography contains, it is an essential contribution to the history of the Viennese Medical School. The book describes the outstanding success of this continental teaching school, and also gives some of the reasons for its later decline. The reader especially concerned with the development of research and with the advances made in this field, will be interested to learn that Starlinger's dogs, deprived of their pyramidal system, were operated on by Wagner-Jauregg. Physiologists will find interesting details about Stricker who was, with Ludwig, a leading experimental physiologist. In conclusion I would like to say that Schönbauer and Jantsch, by editing these memoirs, have done a valuable service to European medical history.

The Unconscious Significance of Hair. By Charles Berg. 1951. London: George Allen & Unwin Ltd. Pp. 106. Price 15*s.*

Psychoanalysts have apparently neglected this promising field, which offers manifest opportunities for discovering sexual symbolism. Dr. Berg brings forward data from the ceremonies of primitive people to support his finding, in patients treated by psychoanalysis, that patting, brushing, shaving, and cutting one's hair is a symptom of deep pregenital conflicts connected with the Oedipus complex and castration, which might otherwise have occasioned serious changes in one's character. Only analysts can judge whether his evidence satisfies them; others will remain sceptical.

Modern Abnormal Psychology. A Symposium. Edited by W. H. Mikesell. 1950. New York: Philosophical Library. Pp. 880. Price $10.00.

This large and expensive book has nothing to recommend it. With few exceptions the contributors fail to say anything new, to organize and express well what they have to say, or to show a mastery of their subject.

Clinical Uses of Intravenous Procaine. By D. J. Graubard and M. C. Peterson. Edited by W. W. Mushin. Oxford: Blackwell Scientific Publications. Illustrated. Pp. 84. Price 8*s.* 6*d.*

This small book assembles the information, previously scattered in a number of individual papers, on a variety of clinical uses for intravenous procaine. The authors themselves have made extensive use of this method of treating various pain-producing states, and have drawn on their own experiences. The main physiological effects of the drug are twofold—production of analgesia and vasodilatation; and from these the therapeutic results

appear to come. Amongst conditions mentioned in which it may prove useful, are allergic reactions, cardiac arrhythmias due to direct cardiac irritation at operation, and a variety of painful lesions including the acute phase of anterior poliomyelitis and post-traumatic pain states. The method as detailed in the book appears to be safe, and is likely to have a wide application in painful conditions of doubtful aetiology which do not respond adequately to routine analgesics. Its vasodilator effect may also prove widely useful in thrombotic and vasospastic conditions.

Incontinence in Old People. By J. C. Brocklehurst. 1951. Edinburgh: E. & S. Livingstone. 62 plates. Pp. 191 Price 30*s*.

This book draws attention to the difficult problem of managing old people who are incontinent. It includes a study of bladder physiology, and demonstrates the troublesome small capacity which these patients often have. The author finds that incontinence may be brought on by insufficient nursing attention to a bedridden patient: once a patient becomes incontinent he is apt to be left out of the bed-pan round, so that no attempt is made to re-train what is lost. The author holds out little hope for increasing the capacity of the bladder in these cases, and yet this seems to be worth further study.

It is also maintained that the bladder becomes spastic in spastic paralysis. It seems likely, however, that diminished capacity in these cases may be due more to a reaction to a period of retention and infection than to any direct effect of long tract disease. For the worst cases, the author has been experimenting with a plaster bed from which the excreta fall into a receptacle. It is hard, however, to believe that this will not cause more difficulties than it will cure. However, though the author's views on treatment are sometimes not very imaginative, this book will be useful to geriatric units and others dealing with the problem. The book is extravagantly produced, and is therefore much too expensive for a work of this kind.

Scientific Thought in the Twentieth Century. Edited by Prof. A. E. Heath. 1951. London: C. A. Watts & Co. Ltd. Pp. 387. Price 42*s*.

This is a symposium with articles written mainly by acknowledged authorities on the subjects treated. Of the 14 articles, eight (zoology, genetics, general medicine, social medicine, neurology, psychology, social anthropology and sociology) deal with aspects of biological science, using the term in its widest sense, and three of these deal directly with sociological problems, while a sociological viewpoint forms an important part of a further three (genetics, general medicine and psychology). This is probably a fair representation of the growing realization in this century of the importance of man's reactions to his fellow men.

The aim of this work is presumably to present to the intelligent amateur both the achievements and the trends of scientific thought which distinguish our times. It is difficult to use the symposium method as a means to this end: to be successful, contributions must be strung together like beads in a necklace. In this volume most of the beads are admirable in themselves, but the string is largely absent. The article on genetics by Dr. Ford, and on zoology by Professor Medawar certainly succeed in combining facts and viewpoints, while Professor R. A. Fisher's article on statistics, a development which probably gives a more distinctive twentieth century flavour to science than any other, is somehow disappointing. The article on neurology is a misnomer, for the data discussed in it are almost exclusively psychiatric.

The tendency of science at the moment seems to be towards a fragmentation of knowledge, and this book, with its informative but rather isolated essays, is representative of this tendency. However, its publication is also a recognition of the need for integration and synthesis which has been increasingly expressed in the last decade.

BOOKS RECEIVED

(Review in a later issue is not precluded by notice here of books recently received.)

Textbook of Psychiatry. By Eugen Bleuler. Translated by A. A. Brill. 1951. New York: Dover Publications Inc. 51 illustrations. Pp. XXVI + 635. Price $7.50.

Diseases of the Nervous System. By W. Russell Brain. 1951. London: Oxford University Press. 4th ed. 84 illustrations. Pp. XXII + 1002. Price 42*s*. (Reviewed in this issue.)

Transactions of the Ophthalmological Society of the United Kingdom. 1951. London: J. & A. Churchill. Vol. LXX, Session 1950. Illustrated. Pp. LXIII + 223.

Psychobiologie der Volksseuche Neurose. By Hans Lungwitz. 1951. Berlin: Walter de Gruyter. Pp. 92. Price DM. 6.40.

Co-ordinating Committee on Abstracting and Indexing in the Medical and Biological Sciences. 1951. France: UNESCO. Pp. 92. Price 5*s*.

Die Darstellung von Lymphströmungen im inneren Milieu des Gehirns. By Richard Arwed Pfeifer. 1951. Leipzig: Geest & Portig K.-G. 73 text figures. Pp. VII + 92. Price DM. 10.-.

Neue Ergebnisse über die Angioarchitektonik der Hypophyse. By Richard Arwed Pfeifer. 1951. Leipzig: Geest & Portig K.-G. 45 text figures. Pp. VII + 71. Price DM. 9.50.

Gesetze und Sinn des Traümes. By K. Leonhard. 1951. Stuttgart: Georg Thieme. Pp. VIII + 146. Price DM. 11.70.

Die Hirngeschwülste im Biologischer und Morphologischer Darstellung. By K. J. Zulch. 1951. Leipzig: Johann Ambrosius Barth. Illustrated. Pp. VII + 232. Price D.M. 24.60.

The Hand in Psychological Diagnosis. By Charlotte Wolff. 1951. London: Methuen & Co. Ltd. Illustrated. Pp. XV + 218. Price 32*s.* 6*d.*

Mind, Perception and Science. By W. Russell Brain. 1951. Oxford: Blackwell Scientific Publications. Pp. V + 90. Price 6*s.* (Reviewed in this issue.)

Psychiatrie Sociale de l'Enfant. Conferences recueillies et redigées par C. Koupernik. 1951. Paris: Centre International de l'Enfance. Illustrated. Pp. 535. Price Fr. Frs. 2,500.

Modern Headache Therapy. By Arnold P. Friedman. 1951. London: Henry Kimpton. Pp. 164. Price 28*s.*

Textbook of Abnormal Psychology. By Carney Landis and M. Marjorie Bolles. 1950. New York: MacMillan. Pp. 634. Price 37*s.* 6*d.* (Reviewed in this issue.)

Das Autogene Training (Konzentrative Selbstentspannung). By J. H. Schultz. 1952. Stuttgart: Georg Thieme. 7th ed. 7 text figures. Pp. XVI + 344. Price DM. 28.50.

Die Exekution des Typus und andere kulturpsychopathologische Phänomene. By W. Wagner. 1952. Stuttgart: Georg Thieme. Pp. VIII + 136. Price DM. 15.60.

THE NOVEMBER (1951) ISSUE

The November (1951) issue contains the following papers:

Hereditary Sensory Radicular Neuropathy. By D. Denny-Brown.

Calcification of the Corpus Striatum and Dentate Nuclei Occurring in a Family. By J. Foley.

The Centripetal Pathway from the Bladder and Urethra within the Spinal Cord. By P. W. Nathan and Marion C. Smith.

Retrograde Axonal Changes of the De-Afferentated Nucleus Gracilis Following Mid-Brain Tractotomy. By P. Glees, J. Soler, and R. A. Bailey.

The Localization of Some Acid Phosphatases in Brain Tissue. By D. Naidoo and O. E. Pratt.

Efferent Connexions of the Human Prefrontal Region, with Reference to Fronto-Hypothalamic Pathways. By E. Beck, A. Meyer, and J. Le Beau.

Anomalous Horizontal Lamination of Nerve Cells in the Supragrannular Cortex of an Idiot Brain. By M. C. H, Dodgson.

Brain Abscess: Aspiration, Drainage, or Excision. By O. V. Jooma, J. B. Pennybacker, and G. K. Tutton.

The Diffusion of Substances in the Subarachnoid Spaces. By G. B. Belloni.

Perceptual Patterns during Recovery from General Anaesthesia. By Joseph Jaffe and Morris B. Bender.

Cessation of Dreaming after Brain Injury. By M. E. Humphrey and O. L. Zangwill.

An Investigation of the Psychiatric Aspects of Disseminated Sclerosis. By R. T. C. Pratt.

A number of copies are still available and may be obtained from the Publishing Manager, British Medical Association, Tavistock Square, W.C.1, price 7s. 6d.